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Esophagus

Esophageal anatomy

- It starts at the level of **C6**.
- The cervical part lies behind the trachea.
- The thoracic esophagus lies behind the left main bronchus then behind the pericardial sac. On both sides, it is related to the lung, pleura and vagus nerve.
- It pierces the diaphragm via esophageal hiatus at level of **T10** and then extends a short distance (~2.5 cm) into the abdomen before joining the stomach.
- **Venous drainage** in to the inferior thyroid, azygos, hemiazygos and left gastric veins. Venous anastomosis (Porto-systemic anastomosis) in the sub-mucosa of the lower esophagus permitting blood to drain either towards
  - the azygos veins and hence to the vena cava, or
  - the gastric veins and hence to the liver via the hepatic portal vein.
- **Physiological narrow areas**
  - The cricopharnyngoeus sphincter.
  - Behind the aortic arch and the left main bronchus.
  - At the esophageal hiatus.

Esophagus	*Cervical part*	*Thoracic part*	*Lower part*
Arterial supply	Inferior thyroid arteries	Aortic branches	Left gastric arteries
Lymphatic drainage	Deep cervical LNs	Mediastinal LNs	Celiac LNs
Nerve supply	Recurrent laryngeal nerve	Vagus nerve	*Lower* esophageal sphincter has some degree of autonomy

Dysphagia

Dysphagia means difficulty in swallowing, while odynophagia means painful swallowing.

Causes :

I- Causes in the mouth : Stomatitis - glossitis - neoplasms of the tongue and cheek.

II- Causes in the pharynx :

1. Pharyngitis.
2. Retropharyngeal abscess.
3. Plummer Vinson syndrome.
4. Pharyngeal diverticulum.
5. Post-cricoid carcinoma.

III- Causes in the esophagus :

[A] Mechanical :

1. Causes in the lumen : Foreign bodies.

2. Causes in the wall :

(a) Congenital e.g. atresia.
(b) Traumatic e.g. corrosive or post-operative stricture.
(c) Inflammatory e.g. reflux esophagitis.
(d) Neoplastic e.g. carcinoma.
(e)schatzki's ring: a circular ring in the distal esophagus of unknown etiology, if symptomatic,treated by dilatation

3. Compression from outside : Malignant thyroid, malignant lymph nodes, aortic aneurysm and mediastinal tumors.

[B] Neuromuscular :

1. Achalasia of the cardia.
2. Diffuse esophageal spasm.
3. Paralysis of the glossopharyngeal or vagus nerves.
4. Tetanus.
5. Myasthenia gravis.
6. Hysteria.

Investigations :

1. Chest X-ray.
2. Barium swallow.
3. Pharyngoscopy and esophagoscopy.
4. Manometric studies.
5. CT scan of the chest.

Esophageal atresia

Any newborn presenting with frothy saliva should be considered to have esophageal atresia until proved otherwise.

Incidence : 1: 4000.

Etiology : The condition is due to a defect in the division of the proximal foregut into ventral tracheobronchial tube and a dorsal esophageal tube.

Pathology :

- **Complications :**

1. The saliva and milk will fill the upper pouch then trickle into the trachea causing bronchopneumonia and frothy saliva.
2. As a result of the distal tracheo-esophageal fistula, the acidic gastric juice will regurgitate through the trachea into the lungs causing acid pneumonia which if persists is fatal.

- **Types :** The commonest type of esophageal atresia is proximal blind atresia with distal tracheo-esophageal fistula (85%). Other rare types include atresia without fistula.

Associated anomalies (VACTERL)

- **V**ertebral
- **A**norectal
- **C**ardiac
- **T**racheal
- **E**sophageal
- **R**enal
- **L**imb anomalies

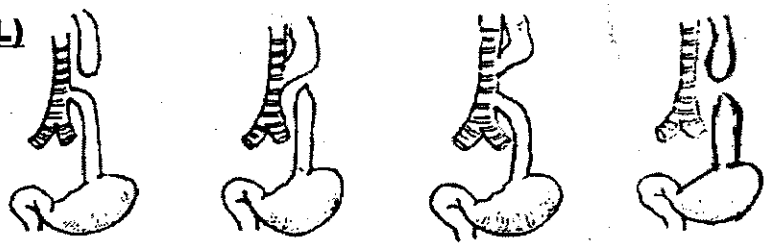


Fig. (1)

Clinical Picture :

I- Antenatal diagnosis :

1. Maternal polyhydramnios.
2. Antenatal ultrasound may show a dilated upper pouch.

II- At birth :

- **General :**
  1. Frothy saliva.
  2. Milk regurgitation (if not diagnosed before feeding).
  3. Attacks of cyanosis.
- **Chest :** Pneumonia.
- **Abdomen :** Abdominal distention with air if the fistula is large or scaphoid abdomen if there is atresia without fistula.

Investigations :

1. Passage of a rigid, radio-opaque (F 10) tube down the esophagus.

2. Plain X-ray of the chest and abdomen after inserting a nasogastric tube. Presence of gastric air bubble will confirm the presence of a fistula.

Treatment :

- **Diagnosis before or immediately after birth is essential.**

1. Atresia with fistula : Right thoracotomy, ligation of the fistula, and primary anastomosis of esophageal ends.

2. Atresia without fistula : Usually there is a long gap, which makes immediate anastomosis difficult, so gastrostomy is done at birth. Further surgery to restore esophageal continuity is to be done months later.

Esophageal injuries

I- Foreign bodies : Can be removed by the rigid or the fiberoptic esophagoscopy.

There are three points of physiological narrowing of the esophagus :

1. The cricopharyngeus sphincter.
2. Behind the aortic arch and the left main stem bronchus.
3. At the esophageal hiatus.

II- Esophageal perforation :

Etiology :

[A] Traumatic :

1. Instrumental : During esophagoscopy or dilatation of a stricture.
2. Foreign bodies.
3. Penetrating or blunt injuries to the neck or the chest.
4. Swallowing corrosives.

[B] Pathological : e.g. carcinoma.

[C] Spontaneous : There is vomiting and incoordinate esophageal motility.The lower esophagus fails to relax in front of the forcibly ejected vomitus. As a result the tear may be :

1. Partial : where the mucosa only is split producing severe bleeding (Mallory-Weiss syndrome).
2. Complete.

Clinical picture :

1. Severe pain
2. The patient is acutely ill with fever, tachycardia and hypotension.
3. Mediastinal emphysema and later subcutaneous emphysema.

Investigations :

1. Plain chest X-ray shows mediastinal emphysema or hydropneumothorax.

2. Gastrograffin swallow shows the site and extent of ruprure.

Treatment :

1. Cervical perforations :

- Nil by mouth, intravenous fluids, drainage of extravasted fluid and intensive antibiotics.

2. Thoracic perforations :

- Early suture of the perforation and chest drainage.
- Late esophagectomy and gastric pull up operation with chest drainage.

Corrosive injury of the esophagus

Etiology : It is a chemical burn due to :
Swallowing of corrosive acids or alkalis (the common example is potash).

Pathology :
- **The extent of damage** depends upon the concentration of the chemical agent and the duration of tissue contact. Corrosive acids are less injurious than alkalis.
- **It is a chemical burn** which may involve the oropharynx, larynx, esophagus, or even the stomach.
- **It may involve** the superficial layers of the esophagus or it may include the whole thickness with even perforation of the esophagus.
- **The injured wall of the esophagus** will be replaced by scar tissue leading to a stricture.

Management of corrosive injuries :

[1] First aid treatment :
1. **Ask the patient to swallow some water or milk.**
2. **Gastric lavage is contraindicated.**
3. **Analgesics, corticosteroids and antibiotics.**
4. **Introduce endotracheal tube** in cases of stridor or dyspnea.

[2] Investigations :
(a) Esophagoscopy : is performed after 24 hours except in patients with suspected perforation.
- The esophagoscope should not be introduced through the damaged area.

(b) Barium swallow : Smooth, long, may be multiple strictures.

[3] Dilatation :
- **Should not be started before the end of the first week.**
- **Criteria of success of dilatation :**

1. Subjective improvement in swallowing.
2. Progressive increase in the diameter of the dilator.
3. The patient is gaining weight.

[4] Surgery :
- **Indications for surgical treatment of the corrosive stricture :**

1. Failure of dilatation.
2. The need for frequent dilatations.
3. If dilatation is hazardous or difficult.

- **A gastrostomy** can improve the patient's condition before definitive surgery.
- Once the patient is fit, **a colon bypass operation** is done.

Neuromuscular abnormalities of the esophagus

(A) Diffuse Esophageal Spasm (Corkscrew Esophagus)

- **In this condition** the normal peristaltic waves are replaced by simultaneous, repetitive and prolonged contraction in response to deglutition.
- **The main symptom** is retrosternal pain rather than dysphagia.
- **Treatment** is by long myotomy from the aortic arch to the cardia.

(B) Achalasia

Definition : This a functional disorder characterized by :

1. Weak peristaltic waves in the body of the esophagus.
2. Failure of the cardiac sphincter to relax during swallowing.

- **The result is a functional obstruction with progressive dilatation of the esophagus.**

Etiology :

Is not exactly known but it is presumed to be due to degeneration of the vagal fibers and the ganglia of the **Auerbach's plexus** of the esophagus itself.

Clinical picture :

1. Incidence : More in the second to the fourth decades. Equal in males and females.

2. Dysphagia : Long standing, slowly progressive, painless which may be more to fluids than solids. At first the dysphagia is intermittent but becomes more constant as the disease progresses.

3. Regurgitation : of foul smelling fluid especially nocturnal.

4. Pulmonary symptoms : as aspiration, wheezing, and chronic cough.

5. The general condition of the patient : is usually reasonable till late.

Investigations :

1. Plain X-ray : may show fluid level in the thorax.

2. Barium swallow :

- Moderate to huge dilatation of the esophagus occurs, which may also show tortuosity. The latter is called **sigmoid esophagus.**
- Smooth rounded termination of the lower end of the esophagus **(Hen's beak).**
- Absence of air in the fundus of the stomach due to continuous stagnation of fluids in the esophagus.

3. Esophagoscopy :

- It reveals a dilated esophagus full of retained food and fluids. The cardia does not relax and may be eccentric in position, and is passed without resistance.
- The main value of esophagoscopy is to exclude the presence of carcinoma.

4. Manometric studies :

- There are weak peristaltic waves in the body of the esophagus after deglutition.
- Failure of relaxation of the cardia in response to swallowing.

Differential diagnosis :

Other causes of dysphagia, esophageal causes (mainly from carcinoma).

Complications :

1. **Pulmonary complications** e.g. pneumonia, chronic cough.
2. **Esophagitis and ulceration.**
3. **Carcinoma :** In the middle one third, of grave prognosis.

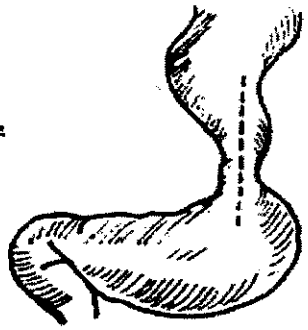
Treatment :

1. Drugs : e.g. Nitrates are of limited clinical value.

2. Forceful dilatation of the cardia by hydrostatic or pneumatic dilators.

A special balloon at the region of the cardia is inflated to produce rupture of the circular muscle fibers to relieve the distal esophageal obstruction.

There is a risk of perforation of the esophagus.



Heller's operation

3. Surgical myotomy (Heller's operation)

= Esophago-Cardiomyotomy :

- Via a thoracic or an abdominal approach.
- The principle is to expose the lower part of the esophagus and cut the muscle fibers completely until the mucosa bulges through the incision.
- Some authors advise the addition of an anti-reflux procedure to prevent reflux esophagitis following the myotomy.

4. Recently, injection of botulinum toxin in the wall of the esophagus at the spastic segment is tried.

Congenital Diaphragmatic Hernia

- **Any newborn presenting with cyanosis and scaphoid abdomen in the absence of heart disease should be considered as having congenital diaphragmatic hernia until proved otherwise.**

Pathology :

- **Types of diaphragmatic hernia :**

1. Posterior or Bochdalek, 90% (left more than right).
2. Anterior or Morgagni.
3. Hiatal hernia (through the esophageal hiatus).
4. Eventration of diaphragm (not a true hernia).

- **Complications :**

1. Ipsilateral lung hypoplasia resulting in pulmonary hypertension.
2. Contralateral lung compression by the abdominal contents.
3. Ischemia of the herniated abdominal contents.

Clinical picture :

1. Respiratory distress, cyanosis & tachypnea.
2. Heart sounds are better heard on the right side in case of the common left sided hernia.
3. Intestinal sounds may be rarely heard on the chest.
4. The abdomen is scaphoid.

Investigations :

1. Antenatal ultrasound is diagnostic.
2. Chest X-ray reveals gas shadow of the stomach or bowel in the thorax.
3. Blood gases.

Treatment :

- **Surgical :**

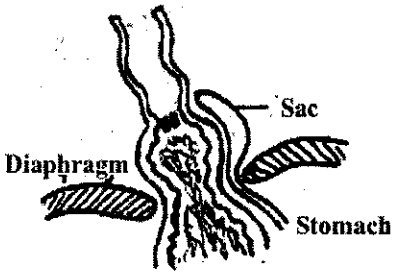
Through an abdominal approach, the herniated abdominal contents are reduced and the defect is closed directly or a mesh is used if the defect is too big.

- **Recently :**

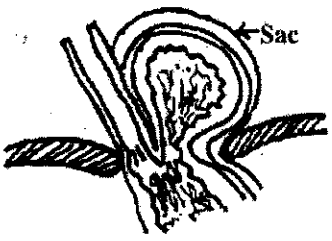
1. Intra-uterine surgical correction was tried.
2. ECMO (Extra Corporeal Membranous Oxygenation).
3. Nitric oxide and toulazoline: they induce pulmonary vasodilatation.

Esophageal Hiatus Hernia

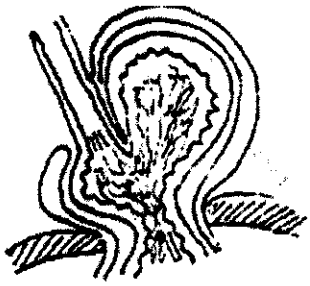
Hernia at the esophageal hiatus may be sliding 85%, paraesophageal 5%, or mixed 10%.



Sliding hernia



Para-esophageal hernia



Mixed hernia

Sliding Hiatus Hernia

- This is due to disturbance in the mechanism responsible for the competency of the lower esophageal sphincter.
- The lower esophageal sphincter is a physiological rather than an anatomical sphincter.
- **The following mechanisms maintain gastro-esophageal competence :**

1. The presence of an intra-abdominal segment of the esophagus.
2. The oblique angle of insertion of the esophagus into the stomach **(The Angle of His).**
3. The pinchcock like action of the fibers of the right crus of the diaphragm.
4. The mucosal rosette of the upper end of the stomach.

Etiology :

1. Increased intra-abdominal pressure as in pregnancy or wearing of tight corsets.
2. Decreased elasticity of the right crus as in obesity and old age.

Pathology :

1. The cardiac orifice and an adjoining part of the stomach herniate through the esophageal hiatus. There is a small empty peritoneal sac on the left side of the stomach.
2. The esophago-gastric junction is no more in the abdomen; consequently, reflux of acid gastric juice to the lower esophagus occurs leading to esophagitis or even ulceration.
3. Reflex esophageal spasm later fibrous stricture may occur.

4. Barrett's esophagus : replacement of esophageal mucosa by columnar epithelium due to prolonged esophagitis.

Clinical picture :

- **Incidence :** Occurs more after the age of forty years and it is more common in females:
- **GERD Syndrome consists of :**

1. Retrosternal discomfort and some **acid regurgitation** especially after heavy meal and lying flat.
2. Pulmonary symptoms.
3. Dysphagia : In the early stages it is due to reflex spasm of the esophagus, but later a fibrous stricture develops in the lower esophagus.
4. Bleeding may occur due to esophagitis or even ulceration.

Investigations :

1. Barium meal in the Trendlenburg's position :

(a) An epiphrenic bulge.
(b) Widening of the esophageal hiatus.
(c) Reflux of barium from the stomach to the esophagus.

2. Esophagoscopy :

(a) Normally the cardia closes on inspiration, but in hiatus hernia it opens.
(b) Reflux of gastric juice through the cardia.
(c) Complications as esophagitis, ulceration and stricture.

3. Manometric studies :

- Loss of normal high-pressure zone at the lower esophagus.

4. PH study (The most important diagnostic test) :

- It will show low PH recording during periods of symptoms.

Treatment :

Conservative measures :

1. Reduction of body weight.
2. Elevation of the head of the bed about 15°.
3. Frequent small meals and avoidance of citrus juices, tomatoes and chocolates.
4. The patient should avoid lifting weights, excessive bending and wearing of tight corsets.
5. Drugs :

(a) Proton pump inhibitors e.g. omeprazole.
(b) H2 blockers e.g. ranitidine.
(c) Antacids relieve hurt burn.

Indications for surgery :

1. Failure of proper medical treatment to control the symptoms.
2. Presence of complications as severe esophagitis, persistent anemia or stricture.
3. Presence of another condition necessitating surgery e.g. duodenal ulcer.

Principles of surgical interference :

1. Reduce the gastro-esophageal junction below the diaphragm and so maintain a segment of the esophagus intra-abdominally.
2. Reduce the size of the esophageal hiatus.

Nissen's fundoplication : depends upon wrapping of lower esophagus by the fundus of the stomach.

Paraesophageal (rolling) hernia

- There is a hernial sac into which the greater curvature of the stomach or even the entire stomach protrudes in the posterior mediastinum.
- The esophago-gastric junction lies below the diaphragm and so reflux esophagitis does not occur.

Clinical picture :

1. **Intermittent dysphagia.**
2. **Post-brandial pain,** which may be confused with angina.
3. **Pressure on the heart** may lead to **cardiac symptoms.**
4. **Occasionally strangulation of the herniated stomach** may occur.

Investigations :

1. Plain X-ray chest : May show the gastric gas shadow in the chest.
2. Barium meal : Will reveal the herniated stomach. The esophago-gastric junction is in its normal location.

Treatment : Only Surgical

- The stomach is retracted downwards, the hernial sac is excised and the hernial defect is closed.

Gastro-oesophageal reflux disease (GORD)

- It is a commonest upper GIT disorder (45% of population)
- Peptic oesophagitis is the commonest endoscopic diagnosis (25% of patients doing upper endoscopy)
- Up to 50 reflux episodes can occur every 24 h.

Mechanisms resisting reflux include;

1. **Competence of the cardiac esophageal junction** due to the effect of the lower oesophageal sphincter (LOS) maintaining a high pressure zone of 15-25 cm H2O physiologically.
2. **Esophageal clearance of the refluxate**
    1. Volume clearance due to normal peristalsis during swallowing.
    2. Chemical clearance by swallows saliva.

Factors promoting reflux and damage

1. **Dysfunction of the cardiac esophageal junction**
    1. Primary weakness of the LOS.
    2. Short length of the intra-abdominal esophagus.
2. **Gastric distention impaired gastric emptying whether organic or functional.**

Clinical picture

1. **Typical symptoms:** heartburn, regurgitation, dysphagia and water brush.
2. **Atypical symptoms**
    a. Chest pain, pulmonary manifestation (simulating bronchial asthma), persistent cough, change in the voice and chocking episodes.
    b. Rarely anemia or hematemesis in case of sever esophageal erosions.

Complications

1. **Variable degrees of erosions and ulcerations.**
2. **Formation of strictures and shortening of the esophagus.**
3. **Barrett's esophagus** (columnar metaplasia)

Investigations

1. **Upper GIT endosopcy.**
    - To evaluate the degree
    - To detect the presence of hiatus hernia
    - To take a biopsy form a suspicious lesion.
2. **Esophageal manometry** to test the pressure of LOS
3. **PH study**

Treatment

I. **Conservative treatment** in the majority of cases as a 1st line of treatment
    a. Weight reduction will markedly improve the symptoms
    b. Stop smoking, alcohol
    c. Avoidance of large fatty spicy acidic meals
    d. Avoidance of recumbency after meals for at least 2 h
    e. Elevation of the head and bed 15 degree to reduce the reflux
    f. Anti-acids, H2 blockers, Proton pump inhibitors and prokinetic drugs

II. **Surgical treatment** ; indicated for
    a. Failure of medical treatment
    b. Development of complications; sever esophagitis, strictures or persistent anemia
    c. Atypical symptoms
    d. Non compliance of the patient

Carcinoma of the Esophagus

Incidence :

- Usually after the age of 50 years.
- More common in males than females, except in the cervical esophagus more common in females.

Predisposing factors :

1. Chronic irritation by certain diets. This may explain the high incidence in certain countries as China & Japan.
2. Excessive alcohol intake and smoking.
3. Corrosive stricture of the esophagus.
4. Achalasia.
5. Long-standing reflux esophagitis.
6. Barrett's esophagus, which is lined by columnar mucosa.

Pathology :

Site : If the cardia is excluded, carcinoma is commonest in the middle third (45-50%) followed by the lower third (33%). The least site is the upper third and cervical esophagus.

Gross appearance :

- **As elsewhere in the gastrointestinal tract, the lesion may present :**

1. An annular scirrhous lesion (the commonest).
2. A malignant ulcer with raised everted edges.
3. A fungating mass.

Microscopic appearance :

1. Squamous cell carcinoma (the commonest) : which may be well differentiated with anaplastic features.

2. Adenocarcinoma may occur in the lower esophagus and is due to :

(a) Carcinoma occurring on top of columnar-cell lined lower esophagus as in Barrett's esophagus.
(b) Upward spread of gastric carcinoma.

Staging :

T_x	Cannot be assessed
T_{is}	Carcinoma in situ
T_0	No evidence of primary tumor
T_1	Tumor less than 5 cm in length
T_2	Tumor > 5 cm
T_3	Any size with extra esophageal spread

N_x	Cannot be assessed
N_0	No clinical palpable LNs
N_1	Mobile unilateral LN affection
N_2	Mobile bilateral LN affection
N_3	Fixed LNs

M_x	Cannot be assessed
M_0	No evidence of distant metastasis
M_1	Distant metastasis

Spread :

1. Direct spread :

- The lesion spreads in both circumferential and longitudinal directions in the wall of the esophagus.
- Malignant cells can be detected in the submucosa many centimeters beyond the palpable edge of the growth.
- Finally, the cells penetrate the muscle layer and the tumor may infiltrate adjacent structures as the trachea, pericardium, pleura, recurrent laryngeal nerves & the stomach.

2. Lymphatic spread :

- According to the site of the lesion, metastases to the draining lymph nodes may occur.
- Lymphatic vessels run longitudinally in the wall of the esophagus and then penetrate the muscle layer to reach the lymph nodes.

(a) The cervical esophagus : Drains into cervical lymph nodes.
(b) The thoracic esophagus : Drains into tracheal, tracheobronchial & posterior mediastinal lymph nodes.
(c) The lower esophagus : Drains into left gastric and celiac lymph nodes.

3. Blood spread : Late.

Clinical picture :

1. Type of patient.

2. Dysphagia :

- Progressive continuous dysphagia is the main symptom.
- At first for solids then for soft diet and lastly for fluids.
- In advanced cases the patient cannot swallow his own saliva leading to continuous drooping of saliva.
- By the time the patient complains of dysphagia about 2/3 of the circumference of the esophagus is involved by the tumor.

3. Pulmonary problems.

4. Severe and progressive loss of weight. In advanced cases the patient is very emaciated.

5. Hematemesis is not a common symptom.

6. Symptoms due to infiltration of adjacent structures as change of voice (recurrent laryngeal nerve) or recurrent chocking due a tracheo-esophageal fistula.

7. On examination : Signs of dissemination as pleural effusion, ascitis, lymph node enlargement, hepatomegaly, and jaundice should be looked for.

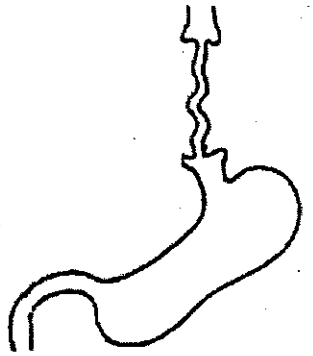
Differential diagnosis :

Other causes of dysphagia, esophageal causes **(mainly from achalasia).**

Investigations :

1. Barium swallow :

- Annular lesions will show an irregular narrowed segment with the characteristic shouldering.
- Cauliflower like lesions will produce persistent irregular filling defects.
- Peristaltic waves may be absent above the lesion due to submucosal infiltration of the esophageal wall.



Submucosal infilteration (shouldering)

2. Esophagoscopy and biopsy.

3. Bronchoscope to detect infiltration of the trachea or bronchi.

4. Plain X-ray chest to detect pulmonary deposits or malignant effusion.

5. Abdominal ultrasound can reveal liver deposits or ascitis.

6. Endoluminal ultrasound can detect invasion of the mediastinum and lymph node enlargement.

7. CT scan can demonstrate the extent of the lesion, and the presence of infiltration of adjacent structures.

Treatment :

Unfortunately many of the patients with esophageal carcinoma are diagnosed when the lesion has reached an advanced stage, and the chance of cure is very low.

I- Inoperable cases :

Criteria of inoperability :

1. **Unfit patients** e.g. with a poor cardio-respiratory status.
2. **Patients with advanced local spread** as those with a tracheo-esophageal fistula, recurrent laryngeal paralysis, and infiltration of the pleura or pericardium.
3. **Extensive lymph node deposits** e.g. supraclavicular lymph nodes, celiac lymph nodes.
4. **Patients with distant metastases** as pulmonary deposits and liver metastases.

- ***They need some palliative treatment to allow them to swallow.***

Methods of palliation :

1. Radiotherapy : A dose of 4500 rads, over 4 weeks. This is particularly suitable for carcinomas of the upper esophagus. Complications of radiotherapy include hemorrhage, perforation and pneumonia.
2. Intubation : The idea is to insert a rigid tube through the stenosed segment, to keep a patent lumen. The tube is either pushed through the lesion by the esophagoscopy or pulled from below after the performance of a gastrotomy. Examples of tubes include the Souttar & Celestin.
3. Palliative surgery : As a palliative colon-by pass operation or gastric pull-up. These are too major operations with a high mortality for a patient with a short life span.
4. Laser photocoagulation.
5. Gastrostomy : This is performed when there is no other alternative. It does not relieve the patient for the inability to swallow his own saliva.

II- Operable cases

[A] Surgery :

- The idea of the operation is to resect the lesion with an adequate safety margin on either side (10 cm) and then to restore the continuity of the gastrointestinal tract.
- **There are many approaches to perform these operations :**

1. Post cricoid carcinoma : either radiotherapy or total laryngeo-pharyngectomy.

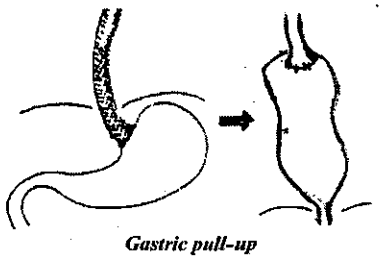
2. Tumors of the lower third of the esophagus : left thoraco-abdominal incision can be used and after excision of the esophagus and the upper portion of the stomach, an esophagogastric anastomosis is performed.

3. Tumors of the middle third (Ivor Lewis) : the stomach is mobilized through a midline incision, then the thorax is opened through the right 5th intercostal space and the esophagus is excised down to the cardia. Then an esophagogastric anastomosis is performed.

4. Many surgeons now adopt the policy of total esophagectomy through a transhiatal approach and pulling the mobilized stomach up to the neck to be anastomosed to the cervical esophagus (gastric pull-up).

- Advantages of this operation :

(a) Guarantee of an adequate safety margin.
(b) The stomach can be mobilized easily.
(c) The esophago-gastric anastomosis is performed in the neck. This is technically easy and if leakage occurs, it will be in the neck and does not lead to mediastinitis.



Gastric pull-up

[B] Radiotherapy : Has a very important role especially in upper and middle third.

Surgical procedures for replacement of the esophagus

1. Gastric pull-up procedures.
2. Colon replacement.
3. Pectoralis major myocutaneous flap.
4. Free jejunal replacement with microvascular anastomosis.

Stomach & Duodenum

Congenital Hypertrophic Pyloric Stenosis (CHPS)

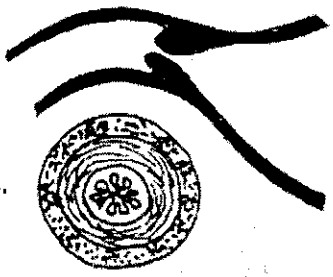
Incidence :

- 3 : 1000
- M : F = 4 : 1.
- More in first born babies.
- More in babies whose mother had pyloric stenosis as a neonate.
- More during spring and autumn.

Etiology :

Unknown, may be genetic or environmental. Muscular factors are probably involved.

Pathology :



- There is gradual progressive hypertrophy of the circular pyloric muscles. With gradual occlusion of the gastric outlet.
- The thickened pylorus fades gradually towards the gastric side and ends abruptly towards its duodenal end.
- The stomach at first hypertrophies to overcome obstruction but later it dilates.

Clinical picture :

Symptoms :

1. Projectile, progressive non-bilious vomiting, classically 2-3 weeks after birth.
2. After vomiting the infant is very hungry.
3. It is usually associated with firm stools and loss of weight.

Signs :

- **General signs of dehydration.**
- **Abdominal :**

1. Test feed : The mother is asked to feed the baby, and then the examiner inspects the abdomen for visible peristalsis passing from left to right.
2. Palpate the abdomen for the olive like pyloric mass (tumor).

Differential diagnosis : From other causes of neonatal vomiting :

1. Gastroenteritis.
2. Gastro-esophageal reflux.
3. Feeding problems.
4. Other obstructive lesions of the gut e.g. intestinal obstruction.

Investigations :

1. Ultrasound : Is the imaging of choice.
2. Barium meal : Is required only if the tumor is not clinically palpable, or is doubtful (10%). It will show dilated obstructed stomach & may show string sign.
3. Laboratory investigations : Serum electrolytes, acid-base balance. It will show Hypokalemia, hyponatremia & alkalosis.

Treatment :

First aid :

1. Correct the dehydration and the electrolyte imbalance.
2. Stop oral feeding.
3. Usually it takes 24 hours to rehydrate the baby to be fit for surgery.

Operation (Ramstedt's operation) :

- The hypertrophied pylorus is grasped between the index and the thumb and a myotomy is performed until the mucosa of the pylorus bulges. If the mucous membrane is injured, the injury should be sutured and reinforced by an omental patch.
- The results of this operation are excellent and the mortality should be around zero.

Ramstedt's operation
Fig. (7)

Peptic ulcer

Definition :

It is an ulcer, which is caused by acid-pepsin digestion of mucous membrane.

Sites :

1. Duodenum (the commonest site).
2. Stomach.
3. The lower esophagus.
4. Jejunum after gastrojejunostomy.
5. Rarely in the small intestine adjacent to a Meckel's diverticulum containing ectopic gastric mucosa.

Etiology :

- **The Gastro-Duodenal mucosal barrier is formed of:**

1. The mucus secreted by the mucosa.
2. The bicarbonate secreted by the mucosa.
3. The rapid repair of mucosal injuries.
4. The abundant mucosal blood flow.
5. The prostaglandins.

- **Factors that weaken the Gastro-Duodenal mucosal barrier:**

1. Drugs e.g. Aspirin & NSAIDs > have Anti-PG effect.
2. Reflux of bile.
3. Helicobacter pylori infection.
4. Ischemia.

- **Results of its disturbance:** Ulceration either acute or chronic.

Acute peptic ulcer

Definition: See before.

Two varieties:

	Multiple erosions	True stress ulcers
Etiology	Non-steroidal anti-inflammatory drugs	Failure of the gastric mucosal barrier due to : Vascular injury with ischemia of mucosa. This may occur in the following conditions : 1. Seen in patients in the ICU. 2. Severe trauma. 3. After major burns. 4. Endotoxic shock.
Pathology	Multiple, shallow and punched out. **Size :** From 1 mm (erosions) to 1 cm or more. **Site :** Occurs in the body and fundus. Usually limited to mucosa & submucosa.	Multiple erosions that if not recognized and treated coalesce to become the condition known as **acute hemorrhagic gastritis.**
Clinically	1. Indigestion (rarely recognized at this stage). 2. Acute upper GIT bleeding.	1. Picture of the cause. 2. Severe upper GIT bleeding.

NB :

- In burns both types of ulcers occur; true stress ulcers **(Curling's ulcer)** occur early in the body or fundus of the stomach while an acute duodenal ulcer occurs late during convalescence.
- It is controversial if **Cushing's ulcer** that occurs after intracranial trauma or operation is true stress ulcer or not.

Diagnosis : Diagnosis is by endoscopy.

Treatment :

"Prophylaxis is better than cure" : H2 blockers, omeprazole, intraluminal antacids and sucralfate have an important role in prophylaxis.

[A] Conservative :

1. Urgent resuscitation.
2. Withdrawing of the offending drug if any.
3. Sucralfate and IV H2 blocking agents.
4. Intraluminal antacids to maintain an intraluminal PH of at least 5 may be effective.
5. Prostaglandins have been tried for their cytoprotective role.

[B] Surgery :

- **Indications :** Failure to control bleeding by conservative measures.
- **Methods :**

1. Total gastrectomy : it has a high morbidity and mortality.
2. Lesser resection procedures : are less effective.

[C] Endoscopy : Recently bleeding may be controlled by :

1. Injection of vasoconstrictors.
2. Laser photocoagulation.
3. Thermal contact.

Chronic peptic ulcer

Duodenal ulcer	Gastric ulcer
Incidence	
Age : 30 - 40 years. **M : F** = 5 : 1. It is decreasing in Egypt. Many younger patients are seen. Ratio of duodenal to gastric ulcer is about 25 : 1.	**Age :** Beyond middle age. **M : F** = 2 : 1.
Etiology	
• Not well resolved. • It is associated with increased gastric acidity. **Which may be due to :** **1. Genetic predisposition :** which produces large parietal cell mass. **2. Increased vagal tone :** shown by the increased amount of overnight gastric acid secretion. May be related to worry and stress. **3. Abnormality of gastrin release and inhibition :** • Gastrin response to a meal is both exaggerated and prolonged. • Defective inhibition by acid. **4. Helicobacter pylori :** was isolated from nearly all duodenal ulcer patients. Eradication of this bacterium markedly decreases the recurrence rate following medical treatment. **5. Hypergastrinemia :** • Pancreatic gastrinoma : Causing the Zollinger Ellison syndrome. • Liver dysfunction. **6. The use of NSAID's.** ***NB.*: smoking will most probably not cause an ulcer, but it may delay its healing.**	• There are **three types** of gastric ulcers : **Type I (60%) :** ulcers occur in the body of the stomach on the lesser curvature. **Type II (20%) :** occur in association with duodenal ulcer. **Type III (20%) :** ulcers occur in the antrum or pyloric canal. • Type II & III are associated with gastric hypersecretion and have the same etiology as duodenal ulcers. • There is a defective gastric mucosal barrier which may be due to : **1. Reflux of bile into the stomach :** causing damage to the gastric mucosal barrier. This allows back diffusion of hydrogen ions, which causes mucosal damage. **2. Antral stasis :** due to defective gastric emptying. **3. Helicobacter pylori.** **4. NSAID's :** particularly if combined with Helicobacter infection. **5. Ischemia :** Evidenced by presence of endarteritis obliterans in the base of the ulcer. **6. Trauma by food.** • It must be remembered that apart from type I&II ulcers, gastric peptic ulcer is not a disease of gastric hypersecretion. However, there must be acid secretion **"no acid no ulcer".** ***NB.*: smoking**, aspirin and alcohol are associated with gastric ulcers. Smoking in particular inhibits ulcer healing.

Duodenal ulcer	Gastric ulcer
Pathology	
Size : Usually small.	**Size :** Larger.
Site : In the 1st part of the duodenum more commonly on the posterior wall and less on the anterior. Sometimes two ulcers occur one on the anterior, the other on the posterior wall, the so-called **"Kissing ulcers"**	**Site :** At the junction of the body and the antrum on the lesser curvature. Some ulcers are saddle-shaped. **(An ulcer situated away from the lesser curvature is highly suspicious of malignancy).**
Edge : Sloping.	**Edge :** Sloping.
Floor : Covered by granulation tissue.	**Floor :** Covered by granulation tissue.
Base : Usually lies in the muscle layer or may even penetrate the duodenum to lie in the pancreas.	**Base :** Usually lies in the muscle layer, or may even penetrate the stomach to lie in pancreas (If posterior) or liver (If anterior) or both (If saddle).
Picture of ulcer at emergency operation (e.g. in bleeding ulcer) : **1. Inspection :** White scar under peritoneal covering. **2. Palpation :** • Localized indurated part of the stomach or duodenum. • Palpating the ulcer crater by finger tip. **3. Special tests :** Gentle rubbing of peritoneal surface by gauze → petechial hemorrhages appear **(stippling sign).**	
Clinical picture	
Type of patient :	
In young active patients, usually well fed.	Usually elderly or old, appear thin & underweight.
1. Pain : The leading symptom.	
Character : Burning.	**Character :** As duodenal ulcer.
Site : In the epigastrium.	**Site :** As duodenal ulcer.
Time : Occurs a few hours after meals.	**Time :** More intimately related to the intake of food.
Nocturnal pain is present.	**No nocturnal or hunger pain.**
Relieved by food and by antacids. Typically these patients carry with them biscuits.	**Relieved by** antacids and vomiting.
Referrence to the back usually indicates penetration of the pancreas.	**Referrence to the back** usually indicates penetration of the pancreas.
Increased by : Spices, hunger.	**Increased by :** Spices.
2. Nausea & vomiting :	
Not prominent features.	Vomiting brings relief and may be self-induced.
3. Periodicity : (pain occurs during periods of activity of the ulcer)	
It is well marked.	Less marked.
4. Appetite :	
Good.	The patient is afraid to eat.
5. Picture of complications.	
6. Physical examination :	
Will only reveal epigastric tenderness. Tenderness differentiates it from malignancy.	

Duodenal ulcer	Gastric ulcer
Investigations	
1. Endoscopy :	
Should be carried out in all patients suffering from typical ulcer dyspepsia.	The principal investigation. Multiple biopsies (at least four) should be obtained from the ulcer.
2. Barium meal : Receded to second place after endoscopy	
Signs of chronic peptic ulcer : 1. Ulcer niche full of barium (in duodenal cap).	**Signs of chronic gastric ulcer :** 1. Ulcer niche full of barium (usually on lesser curvature).
2. Duodenal cap is deformed from spasm and later fibrosis.	2. Notch on greater curvature opposite the ulcer niche due to spasm and later fibrosis of circular muscle fibers.
3. Stomach is steer horn (hypertonic).	3. Stomach is J-shaped (hypotonic).
4. Localized tenderness under screen.	4. Localized tenderness under screen.
5. Complications due to scarring of the ulcer may be seen : e.g. pyloric stenosis.	5. Complications due to scarring of the ulcer may be seen : Hourglass stomach, teapot stomach and pyloric stenosis.
6. After emptying, a flake of barium remains in ulcer niche, and mucosal folds (barium relief) are seen radiating from it.	After emptying, a flake of barium remains in ulcer niche, and mucosal folds (barium relief) are seen radiating from it.
3. Investigations to detect Helicobacter pylori :	
➢ ***serology*** ➢ ***Gastric biopsy smearing and culture*** on skirrow's medium &identified by morphology, biochemical reactions (urease +ve) and sensitivity to antibiotics. ➢ ***Urease test:*** specimen is placed onto a medium containing urea with color indicator; the urease will split the urea and will change the PH and the color. ➢ ***Urea breath test:*** patient drinks urea with radioactive C14,release of CO2 containing C14 indicates splitting of urea by H.pylori	

Zollinger Ellison's syndrome (Gastrinoma): 2 types are recognized :

1. ZES type I : Rare, there is antral cell hyperplasia → High gastrin level with chronic DU.

2. ZES type II: It is a non B-cell tumor of pancreas secreting gastrin. It is a malignant tumor in about 60 % of cases. There are intractable peptic ulceration (often in jejunum), hypergastrinemia with massive acid hypersecretion. It may be part of **MEN I.**

Investigations :

- Radioimmunoassay estimation of serum gastrin shows serum levels more than 500ng/L.
- Gastric function tests and estimation of circulating gastrin.
- **SRS**(somatostatin radioisotope scan) : can localize gastrin producing tumors.

Treatment :

1. Type I: Partial gastrectomy to remove antral mucosa.

2. Type II: If a tumor is seen, resection is advised. **But**

- Only 20% of cases are resectable because of multiplicity and poor localization.
- Usually resection is impossible, so total gastrectomy is done to completely abolish gastric secretion.
- A less effective therapy is intensive therapy with P.P.Is.

NB.:***ulcers are suspected to be due to Z-E syndrome*** with any of the following conditions:
1. Resistant to treatment. 2. Recurrent. 3. Multiple 4.in unusual sites. 5. at extremes of age.

{A} Treatment of chronic duodenal ulcer

I- Medical

The aim : Is to reduce the acidity to allow the ulcer to heal and maintain its healing.

1- General measures :

1. Physical and mental rest.
2. Diet : Bland diet, avoid irritant foods or drugs.
3. Milk gives some relief due to its neutralizing effect, but it is a potent stimulant to HCL.

2. H_2 receptor blockers : Such as :

- Cimitidine 800 mg at bedtime **OR** Ranitidine 150 mg twice daily **OR** Famotidine 40 mg at bedtime.
- The ulcer heals within 6 weeks but full dose treatment is continued for a few weeks more.
- Endoscopy is repeated to evaluate healing.
- The main disadvantage is the high rate of recurrence.

3. More recently a proton pump blocker :

- It reduces gastric acidity to zero in a matter of days.
- Given 30 minutes before meals with duration of action exceeding 24 hrs.

1. **Omeprazole** 20-40mg/d.
2. **Lansoprazole** 15-60mg/d.

- **Disadvantages of long term therapy with 2 or 3 is :**

1. Expensive.
2. Prolonged gastrinemia : Gastrin is a trophic hormone to the gastric mucosa.
3. Omeprazole often results in duodenal bacterial overgrowth.
4. Gastric neoplasia is at least a theoretical risk.

4. The treatment of Helicobacter pylori :

A triple drug therapy consisting of **metronidazole**, **tetracycline** or **amoxicyllin**, and **bisthmus compounds** given for 2 weeks gives excellent results in 90% of cases.

N.B:
H.pylori is a microaerophilic gram negative, spiral shaped and flagellated organism, present in almost half of the world population and ***it causes*** chronic gastritis, peptic ulceration and gastric lymphomas.

5. Repeated small doses of antacids : Can help in the rapid relief of pain.

6. Agents that enhance mucosal resistance :

1. **Sucralfate :** 1g/6hrs stimulates mucus, bicarbonate & prostaglandin production.
2. **Misoprostol :** Prostaglandin analogue.
3. **Antacids.**

Two suggested solutions to the problem of high recurrence rate after medical treatment :
1. Interval therapy : It consists of giving full dose regimen at the time of maximum stress, which is the period when the ulcer activity is at the greatest risk of recurence.
2. Another way is to give a small dose at bedtime for a prolonged period.

II- Surgery

Indications : Failure of medical treatment to control symptoms.

1. **Relapses** after repeated attempts of adequate medical treatment.
2. **Financial factors.** If the patient cannot afford medical treatment.
3. **Bad patient compliance :** Many patients are unwilling to take tablets or other medications for a long time and will probably miss many doses.
4. **Occurrence of complications** e.g. pyloric stenosis.

The aim : Is to achieve permanent and adequate reduction of gastric acid secretion by either :

1. Abolishing the nervous phase of gastric acid secretion by vagotomy which may be :

- Truncal. • Selective. • Highly selective. • Seromyotomy.

2. Abolishing the hormonal phase of gastric acid secretion by antrectomy : Some surgeons combine vagotomy and antrectomy.

3. Reduction of the parietal cell mass by a subtotal gastrectomy : Only of historical interest.

1- Truncal vagotomy

- 80% reduction of gastric HCL secretion (by the passage of time this decreases to about 50%).
- Recurrence rate of about 5-10%.

Side effects :

1. Denervation of the stomach can be compensated for by performing a drainage procedure e.g. pyloroplasty or gastrojejunostomy.
2. Denervation of the entire abdomen except for the hindgut.

2- Selective vagotomy

- Denervate the stomach only by cutting anterior vagus below the hepatic branch and posterior vagus below the celiac branch.
- A drainage procedure is still required.
- It is now rarely if at all performed.

Gastric drainage procedures :

1- Gastrojejunostomy.

2- Pyloroplasty. This operation destroys the pyloric sphincter by cutting it. It has many types the most popular is the **Heineke-Mikulicz type** where the pylorus is divided longitudinally and sutured transversely.

3- Highly selective vagotomy
(Pareital cell vagotomy = proximal gastric vagotomy)

It is designed to denervate only the body and fundus of the stomach i.e. the acid secreting parts of the stomach and stopping short of the branches of the antrum **(the crow's foot)** thus preserving gastric emptying and avoiding the drainage procedure.

4- Lesser curve seromyotomy

- Gives exactly the same denervation as proximal gastric vagotomy.
- Most surgeons perform it anteriorly only adding a posterior truncal vagotomy.

Antrectomy :

- It is designed to remove the pyloric antrum, which is the site of production of gastrin hormone.

(In this country the two most popular operations for duodenal peptic ulcer are truncal vagotomy and gastrojejunostomy and proximal gastric vagotomy).

{B} Treatment of chronic gastric ulcer

[A] Conservative : As chronic duodenal ulcer with the following considerations :

1. Because of the risk of being malignant, it is essential to exclude malignancy before deciding medical treatment.

2. Endoscopy should be carried out after 6 weeks regardless of symptomatic improvement.

(a) If the ulcer shows no attempt at healing, it should be considered malignant and surgery performed.

(b) If partial healing is seen continue medical treatment for another 2 months (never more than 6 months) and repeat endoscopy **(it should be noticed that some patients with gastric cancer may improve on medical treatment).**

[B] Surgery :

Indications :

1. Failure of healing to start at 6 weeks or of complete healing at 6 months after medical treatment.
2. Suspicious of malignancy.
3. Bad patient compliance and financial factors.
4. Occurrence of complications.
5. Some surgeons regard gastric ulcer as an indication for surgery without any attempt at medical treatment.

The aim : Is to remove the ulcer or the entire ulcer bearing area.

Methods :

1. Partial gastrectomy : Which removes the ulcer together with the entire ulcer bearing area. The operation is ended by gastroduodenal anastomosis **(Billroth I)**, which is the standard procedure, or by a gastrojejunostomy **(Billroth II or polya gastrectomy operation).**

2. Truncal vagotomy with a drainage procedure plus resection of the ulcer.

3. Highly selective vagotomy and resection of the ulcer.

- **In this country, a form of gastrectomy remains the standard surgical treatment of benign gastric ulcer.**

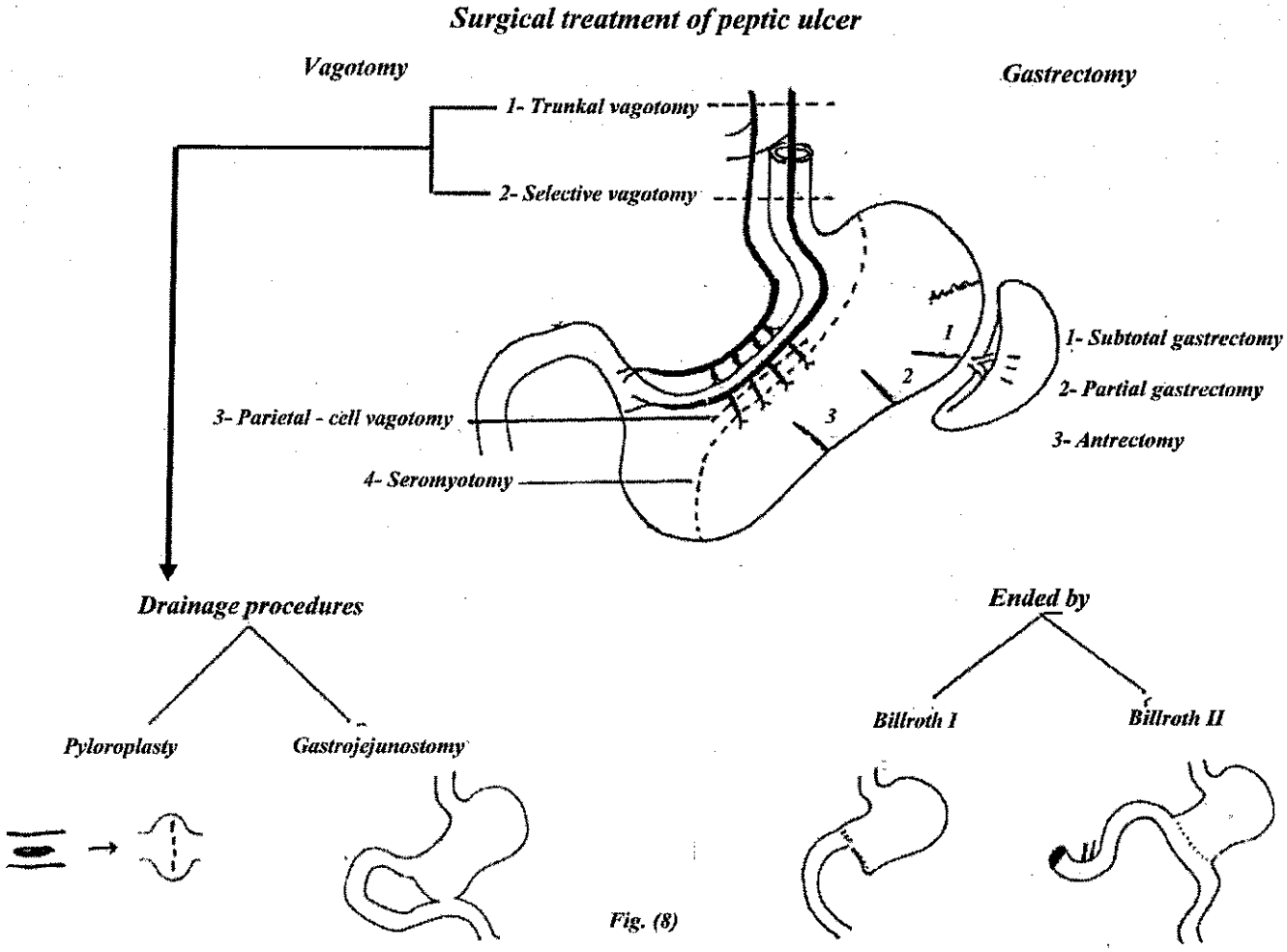


Fig. (8)

Complications of peptic ulcer

These are :

1- Acute complications : bleeding and perforation : Occur during periods of activity of the ulcer.

2- Chronic complications : Penetration, pyloric stenosis, Hourglass and tea pot stomach, they occur insidiously.

I - Perforated peptic ulcer

(A) Acute perforated peptic ulcer

Incidence :

- Anteriorly situated ulcers perforate more commonly.
- Duodenal ulcer perforates more commonly than gastric ulcer.

Exciting causes :

- During a relapse precipitated by alcohol, irritants, nervousness, etc.
- In 20% of the cases perforation is the first presentation.

Pathology	Clinical picture
[A] Stage of perforation	**[A] Stage of shock**
• Yielding of the ulcer base pouring gastric contents into the peritoneal cavity.	• **Usually short and the patient is not usually seen in it.** 1. Sudden severe upper abdominal pain. 2. Vomiting may occur once. 3. Neurogenic shock. 4. Upper abdomen shows tenderness and board like rigidity.
[B] Stage of chemical peritonitis	**[B] Stage of lucid interval**
• Peritoneum will react by the production of serous alkaline fluid, neutralizing the acid and bringing antibodies to hinder the growth of bacteria temporarily. • **The length of this stage depends on :** 1. Size of the perforation. 2. The contents : Longer duration with empty stomach. 3. The general resistance of the patient. • **Duodenal ulcer perforation is less serious, with a longer lucid interval because:** 1. Contents are more sterile. 2. Contents are less irritant. 3. Smaller size. **4.** Poured out contents flow along the Rt paracolic gutter and tend to localize in Rt iliac fossa. Gastric ulcer perforates directly into the general peritoneal cavity producing generalized peritonitis.	• Patient is relieved from shock, and vital signs improve. • Pain diminishes and patient comes walking to the hospital. **On examination :** 1. Spreading tenderness and rigidity towards the abdomen. 2. Obliteration of the liver dullness. 3. Shifting dullness may be present.
[C] Stage of septic peritonitis	**[C] Stage of septic peritonitis**
• Occurs when body resistance fails. • Microorganisms invade the peritoneal cavity and pus forms.	1. Vomiting, distention, dead silent abdomen & absolute constipation. 2. Hypovolemic shock sets in and becomes more profound. 3. Patient dies in few days if untreated.

Differential diagnosis :

Perforation has to be differentiated from perforated acute appendicitis, acute cholecystitis, acute pancreatitis and intestinal obstruction. At the onset it has to be differentiated from myocardial infarction.

Investigations :

1. Laboratory : Electrolytes - blood picture (leucocytosis).
2. X-ray in the erect position : will reveal air under the copula of the diaphragm.
3. Peritoneal tapping : will reveal bile.
4. Gastrograffin meal : If perforation is suspected and plain X-ray failed to reveal gas under diaphragm.

Treatment : Urgent Operation

Preoperative preparation and resuscitation :

1. A Ryle's tube with continuous aspiration.
2. IV fluids are set guided by electrolyte and PH estimations.
3. IV antibiotics are given.
4. IV H2 receptors blocking agents are given.
5. Careful attention to respiration.
6. Late cases with toxemia require blood transfusion.

At operation : The simplest procedure is done :

1. Closure of the perforation with a covering omental patch.
2. Thorough peritoneal toilet.
3. The abdomen is closed with drainage.
4. In the case of a perforated gastric ulcer a biopsy must be obtained.

Postoperative :

The patients are treated medically for their ulcer disease.

(B) Subacute perforation

This is a small perforation allowing only a minimal amount of contents to enter the peritoneal cavity and is rapidly sealed. It gives the same early signs but much reduced. If diagnosed correctly it can be treated conservatively.

(C) Chronic perforation

Means penetration into adjacent structures as the pancreas. It is suspected when the pain becomes more or less persistent and referred to the back.

II- Bleeding

Predisposing causes :

- Factors causing exacerbation of an ulcer.
- Occasionally bleeding is the first presentation of the ulcer.

Bleeding may be :

1. On a microscopic level : Considered as a part of the disease process.
2. Mild : From granulation tissue in the floor of the ulcer.
3. Moderate : Erosion of a small vessel in the floor of an ulcer.
4. Severe : Due to penetration of a large extragastric vessel as the gastroduodenal artery. This hemorrhage is usually fatal.

Clinical picture :

1. Hematemesis : Dark coffee ground due to acid hematin.
2. Melena : Altered blood in stools.
3. Manifestations of progressive shock.
4. Signs :

(a) Exclude false hematemesis by examination of the nose and pharynx.
(b) Exclude portal hypertension as a cause of hematemesis, the commonest cause of hematemesis in Egypt being bleeding varices.

Investigations :

1. Hemoglobin and hematocrit : Useful only after a few hours when hemodilution occurs.

2. Endoscopy : When the general condition of the patient is stabilized.

Differential diagnosis : Of hematemesis.

Treatment :

- Most of these cases stop bleeding spontaneously.
- Conservative treatment should always be considered whenever possible.

[A] Conservative treatment :

- **It has been said that conservative treatment of a bleeding peptic ulcer should be carried out at the door of the operating room for the rapidly changing nature of the clinical course.**

1. Absolute rest in bed under opiate sedation.
2. Correction of hypovolemia by IV crystalloids and blood transfusion (Fresh blood in sufficient amount is the best single item to resuscitate and promote arrest of bleeding).
3. Careful monitoring of pulse, blood pressure and hematocrit.
4. IV H2 receptor blockers or omeprazole.
5. Antacids should be given frequently and liberally.
6. Cold drinks and bland food should be given.
7. Lavage of the stomach by cold saline is recommended by some surgeons.

[B] Endoscopic treatment :

- The ulcer is usually covered by a blood clot or there may be an actual spurting artery.
- Gastroscopy provides a diagnosis of the cause of bleeding and can also achieve hemostasis.
- **Indications of endoscopic hemostasis :**

1. The ulcer is seen to be actively bleeding.
2. Stigmata of recent bleeding because they indicate likelihood for rebleeding.
   (a) A visible vessel in the ulcer base.
   (b) Fresh clot covering the ulcer.

- **Methods of endoscopic hemostasis :** Fine instruments are introduced through the channel of the gastroscope, reach the ulcer and arrest bleeding by either laser coagulation, thermal coagulation, diathermy coagulation, or by injection of the ulcer base by adrenaline or alcohol.

[C] Operative treatment :

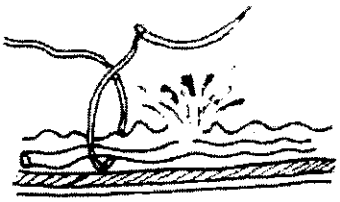
Indications :

- **Absolute in :**

1. Patient under adequate medical treatment.
4. If bleeding recurs while the patient is still in hospital.
3. If bleeding continues as evidenced by the need to transfuse 1000 ml or more of blood per day to maintain stability.
4. If the initial bleed was severe (2000 ml or more).

- **Relative in :**

5. Old arteriosclerotic patients:
   (a) They cannot withstand the ill effects of shock.
   (b) Their arteries are also incapable of retraction enough to stop hemorrhage.
6. Long history of ulcer diseases.



The decision to operate should be taken early (within 48-72 hours), as the results of late surgery are poor with a high mortality :

(A) For bleeding gastric ulcer :

- Gastrotomy and under running the ulcer (fig 9) together with a biopsy or resection combined with vagotomy and drainage is more popular.
- The classical management is gastrectomy, but it has a significant morbidity and mortality.

(B) For bleeding duodenal ulcer :

- Under running the ulcer.
- Pyloroplasty with trunkal vagotomy is the standard treatment.

III- Fibrous Contractures of Peptic Ulceration

These are :

1. Pyloric stenosis.
2. Hourglass stomach.
3. Teapot or handbag stomach.

- **They are complications of healing ulcers rather than complications of their activity.**

Pyloric stenosis

Pathology :

1. Pylorus : Cicatrized and stenosed, showing the specific pathology.
2. Stomach : At first hypertrophied then dilated may reach down to the pelvis.
- The mucosa : At first it shows hypertrophic gastritis, then atrophic gastritis and true hypoacidity, due to stagnation and fermentation.

3. Intestine : Normal and collapsed.

Complications : Due to repeated vomiting :

1. **Metabolic alkalosis** and may be **tetany.**
2. **Hyponatremia** (Na loss) and **hypokalemia** (K loss).
3. **Dehydration, loss of weight** and **avitaminosis.**
4. **Antral stasis** releases gastrin leading to other ulcers.
5. **Respiratory tract infection** from inhalation of vomitus.

Clinical picture :

1. **Long history of ulcer dyspepsia,** or it may be the first presentation.
2. **Vomiting :** Projectile, not bile stained, contains food of previous meals or days, foul odor revealing fermentation (characteristically in the evening).
3. **The initial periodic ulcer pain is lost** and replaced by **epigastric discomfort and fullness.**
4. **Progressive constipation.**
5. **Picture of complications.**
6. **Signs :**

(a) General : Usually under weight. See complications.
(b) Abdominal :

- Epigastric fullness (may show the contour of the enlarged stomach).
- Succussion splash can be heared.
- Peristaltic waves propagating from left to right.

Differential diagnosis :

1. Congenital : Infantile hypertrophic pyloric stenosis **(not to be mentioned in peptic ulcer).**
2. Traumatic : Impacted F.B., corrosive ingestion, after pyloroplasty.
3. Inflammatory : The commonest being a cicatrized duodenal or prepyloric gastric ulcer. Less commonly TB, Crohn's disease, Syphilis etc.
4. Neoplastic : Cancer pylorus. Less commonly sarcoma or leiomyoma.
5. Pressure from outside : Lymph nodes, bands, tumors of head of pancreas.
6. Functional : Pylorospasm or achalasia.

(The commonest cause being duodenal ulcer and cancer pylorus).

Investigations :

1. Estimation of hemoglobin and hematocrit, serum electrolytes.

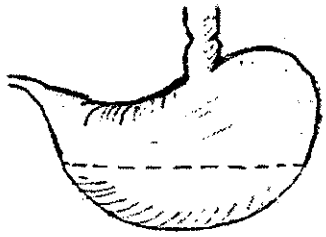
2. Endoscopy : Pyloric ring is inactive and will not pass the tip of the endoscope. The main value is to detect the presence of a tumor at the pylorus to obtain biopsies from it.

3. A barium meal will show :

- Dilated stomach often reaching the pelvis.

(Soup dish appearance).

- Delayed gastric emptying.
- Irregular filling defect in case of carcinoma, however it will often miss a small pyloric carcinoma.



Soup dish

Management :

Pyloric stenosis requires surgery, which is not an emergency and needs **Proper Preoperative Preparation :**

1. The stomach should be sucked and washed through a nasogastric tube.
2. Correction of fluid, electrolytes and acid-base balance.
3. A high protein fluid diet should be given and is supplemented by intravenous alimentation.
4. Parenteral vitamins.
5. Blood transfusion of packed cells to correct anemia.
6. Chest physiotherapy and antibodies.
7. Some cases of pyloric stenosis occur due to muscle spasm and a marked inflammatory reaction around an active duodenal ulcer. These cases respond well to medical treatment.

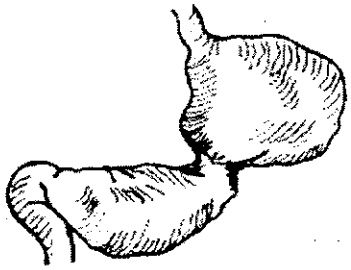
Surgery :

- Truncal vagotomy and gastrojejunostomy is the standard treatment for the condition.
- Other options :

1. Gastrojejunostomy alone may be done in elderly or unfit patients.
2. Endoscopic balloon dilatation.
3. Partial gastrectomy + gastrojejunostomy.

Hourglass stomach

Due to : Cicatrized saddle shaped ulcer on the lesser curvature.
Clinical picture : Symptoms of gastric obstruction.
Differential diagnosis : Carcinoma.
Investigations : Endoscopy and a barium meal.
Treatment : Partial gastrectomy.



Hour glass stomach

Teapot stomach

Due to : Cicatrized ulcer on the lesser curvature with shortening of the lesser curvature.
Clinical picture : Of obstruction as the pylorus is higher and not at a good position for the stomach to empty through it.

IV- Malignancy

- Duodenal ulcer never turns malignant.
- Gastric ulcers very rarely turn malignant (less than 1% of cases).
- The clinical problem, however, is usually not whether a gastric ulcer has turned malignant or not, but whether an ulcer is malignant or not.

V- Complications of Gastric Operations

[A] General complications.
[B] Specific complications as :

Early complications :

[A] Bleeding from the anastomotic line.
[B] Stomal obstruction, due to :
1. Edema at the stoma.
2. Technical error.
3. Retrograde jejunogastric intussusception.
4. After vagotomy in which the stoma is actually patent but the stomach is atonic and does not empty.

[C] Duodenal blowout :
- Follows Billroth II anastomosis.
- Usually occurs around the fourth day.
- Very serious complications due to excessive fluid and electrolyte loss.

[D] Postoperative pancreatitis.

Late complications :

[A] Recurrent ulceration :

Etiology :
1. Inadequate surgery e.g.
 (a) Missing a vagal trunk, usually the posterior.
 (b) Missing the so-called **"criminal nerve"** which is a branch of the vagus nerve running over the lower esophagus.
 (c) Leaving part of the gastric antrum with the duodenum in gastrectomy.
2. Other causes such as a gastrinoma causing the Zollinger-Ellison's syndrome.

Site :
1. Stomal i.e. on the anastomotic line. 2. Gastric. 3. Jejunal. 4. True recurrence of the original ulcer.

Clinical picture : Recurrence of ulcer symptoms.
Investigations : Endoscopy.
Treatment :
1. H_2 receptor blockers or omeprazole.
2. Re-operation :
 (a) Following vagotomy : An antrectomy is performed.
 (b) Following gastrectomy : A vagotomy is performed.

[B] Dumping :

Definition : "Syndrome with vasomotor and gastrointestinal symptoms following meals".
- If symptoms occur within the first ½ hour after meals it is known as early dumping.
- If delayed for about 2-3 hours it is called late dumping.

1. Early dumping :

Clinical picture :
- Gastrointestinal symptoms : Epigastric fullness and pain with nausea and ending in explosive diarrhea.
- Vasomotor symptoms : Sense of weakness, flushing and palpitation.

Pathogenesis :
- Rapid gastric emptying with the delivery of a hyperosmolar solution to the proximal small gut with the result of shift of fluid from the circulatory plasma to the lumen of the proximal small gut.

Treatment :
- Prevention, small frequent feeds rich in proteins and fats and less in carbohydrates.
- Most cases are controlled by assuming the supine position.
- Operation is rarely required e.g. interposition of a reversed loop between the stomach and small intestine.

2. Late dumping :

Clinical picture : Picture of hypoglycemia, sweating, palpitation, tremors and confusion, which are relieved by carbohydrate ingestion.

Pathogenesis : Overshot of insulin which is caused by rapid delivery of large amounts of carbohydrates to the small intestine.

Prevention : By giving frequent small feeds of a carbohydrate low diet.

[C] Biliary gastritis (Alkaline reflux gastritis).

[D] Afferent loop syndrome :

- **Due to** obstruction of the too long afferent loop.
- **Clinical picture :** fullness and epigastric pain following meals, followed by projectile bilious vomiting.
- **Explanation :** Food has stimulated pancreatic, and bile secretion, which distend the afferent loop. Pressure increases until obstruction is overcome and the loop empties itself into the stomach.
- **Treatment :** Operative treatment is essential and consists of conversion of the anastomosis to a Roux-en-Y loop.

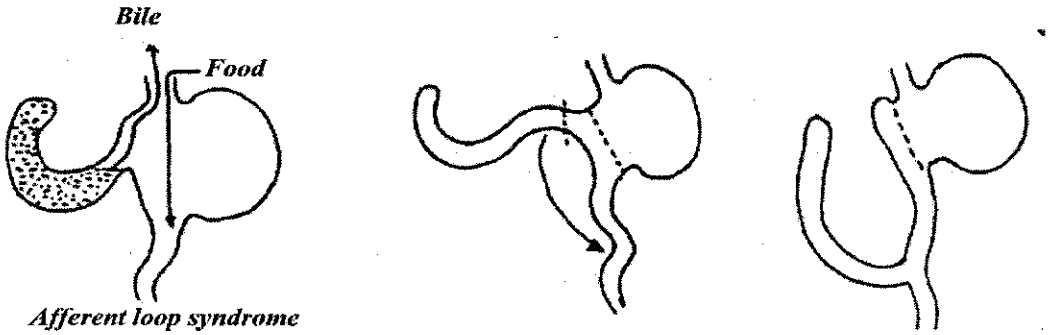


Fig. (12)

[E] Post-vagotomy diarrhea :

Etiology : Unknown. The condition gets better as time goes on.

[F] Gastrojejunocolic fistula :

Due to a recurrent ulcer following gastrojejunostomy.

Clinical picture : Severe diarrhea, ill health, loss of weight and anemia (the diarrhea is due to enteritis caused by the entry of colonic contents into the jejunum).

Investigations : for its visualization a barium enema (and not meal) is needed.

Treatment : Surgical. Proximal diverting colostomy is performed at first, followed later by correction of the fistula.

[G] Post-gastrectomy nutritional disturbances :

1. Weight loss.

2. Steatorrhea and diarrhea : Due to lack of mixing of food with pancreatic and biliary secretions.

3. Iron deficiency anemia : Daily iron supplement.

4. Megaloblastic anemia : Intramuscular vitamin B12 injections.

5. Vitamin B deficiency : Regular prophylactic daily supplement.

6. Calcium deficiency : Due to reduced acidity.

[H] Increased risk of stomach cancer following both partial gastrectomy and vagotomy.

Neoplasms of the stomach

Benign Gastric Neoplasms

- The commonest is the **leiomyoma.**
- **Clinical picture :** Vague symptoms or epigastric mass.
- **Investigations :** Smooth filling defect in a barium meal.
- **Complications :** Bleeding, malignant transformation.

Carcinoma of the Stomach

Except in Japan and despite the great advances that have occurred in surgery, radiotherapy, and chemotherapy, the prognosis of gastric cancer has remained very bad. The overall 5 years survival rate is about 5%.

Incidence :

- Falling steadily throughout the world except in Japan.
- **Sex :** More in males.
- **Age :** Above 40 years.

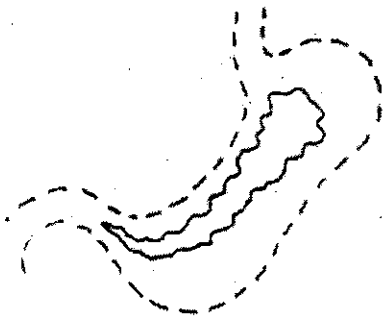
Etiology :

1. Dietary : Any factor that causes gastritis can cause carcinoma e.g. tobacco, alcohol, spices and increased salt intake.
2. Achlorhydria : The presence of nitrates in food leads to the production of N-nitrosamines by the action of bacteria in the achlorhydric stomach.
3. Pernicious anemia.
4. Helicobacter pylori (not sure).
5. Genetic predisposition : Persons with blood group A are more susceptible.
6. Benign gastric ulcers : very rarely turn malignant (see before).
7. Benign gastric neoplasms : as gastric polyps.
8. Following gastrectomy : This is due to biliary reflux gastritis.

Pathology :

Site :

- **Pyloric antrum** 65%.
- **Linitis plastica :** The least.



Linitis plastica

Macroscopic picture :

[A] Classic Description :

1. A fungating cauliflower like mass.
2. An ulcer with raised indurated edges.
3. Colloid carcinoma.
4. The diffusely infiltrating variety "Linitis plastica" :
   - The wall of the stomach is greatly thickened and indurated while the lumen is greatly reduced.
   - May occur only in the antrum or may affect the stomach more diffusely.
   - The mucous membrane is intact and it may be missed by endoscopy.

The classical description is now known as late cancer of the stomach.

[B] Recent Description :

- Screening program for stomach cancer that lead to the discovery of early cases.
- The exact definition of which is **"a carcinoma limited to the mucous membrane or the submucosa".**
- **Three types are described :**

1. Protruding. 2. Superficial. 3. Excavated (penetrating).

- Unfortunately, in this country and in most others, all cases are late cancers.

Microscopic picture :

- Adenocarcinoma with various grades of differentiation.
- A colloid carcinoma carries a bad prognosis.

Staging :

T_{is}	Carcinoma in situ
T_1	Tumor confined to mucosa
T_2	Tumor involves submucosa
T_3	Tumor penetrates serosa
T_4	Diffuse involvement of stomach wall or extra-gastric spread
N_0	No lymph node metastasis
N_1	Perigastric lymph nodes within 3 cm of gastric wall
N_2	Lymph nodes > 3 cm from gastric wall
N_3	Involvement of central abdominal lymph nodes (celiac)
M_0	No metastases
M_1	Distant metastases

Spread :

1. Direct spread : Infiltration of the wall of the stomach in depth, in circumference, and in length.

- Infiltration of neighboring structures as the liver, spleen, pancreas or colon.

2. Lymphatic spread : Both by permeation and by embolization.

(a) The proximal half of the stomach drains to the left gastric lymph nodes and splenic lymph nodes.
(b) The antrum drains into the right gastric and subpyloric lymph nodes.
(c) The greater curvature drains into gastroepiploic nodes.

- **From a, b & c lymphatic drainage pass to celiac or superior mesenteric lymph nodes.**

3. Blood stream spread : Occurs mainly to the liver and very rarely to bones.

4. Transcelomic spread :

(a) To the ovaries in younger females **(Krukenberg's tumor).**
(b) To the Douglas pouch lead to what is known as **Plummer's shelf.**

Clinical features :

This disease is usually diagnosed at a late stage because of the vague and often mild symptoms it gives.

- **It may present by :**

[1] Dyspepsia group : A person above 40 years who starts to complain of dyspepsia should be fully investigated for the possibility of stomach cancer. It differs from chronic gastric ulcer in :

1. No periodicity.
2. Pain is more severe and continuous.
3. Anorexia.
4. Vomiting does not relieve pain.

[2] Insidious group : Vague symptom of : **Anemia** (pallor and easy fatigability). **Asthenia** (wasting and weakness). **Anorexia** (especially for meat). Anemia in the elderly should always raise the possibility of malignancy, the stomach being one of the commonest to cause it.

[3] Obstructive type :

1. Tumors at cardia produce dysphagia.
2. Tumors at pylorus produce malignant pyloric obstruction.

[4] Mass group : Epigastric mass (for D.D.).

[5] Metastatic group : A hard irregular liver due to secondaries, jaundice, malignant ascitis, or an enlarged left supraclavicular lymph nodes **"Troisier's sign",** all of which are signs of inoperability.

[6] Other presentations :

1. Hematemesis and melena (uncommon).
2. A patient known to have a gastric peptic ulcer who becomes resistant to treatment.
3. Superficial thrombophlebitis migrans **(Trousseau's sign).**

D.D.:

[A] Causes of dyspepsia : e.g. biliary.

[B] Causes of mass in the epigastrium :

1. Gastric tumors.
2. Transverse colon : Carcinoma, intussusception.
3. Small intestine : Intussusception, lymphoma.
4. Left Lobe of liver : Tumor or cyst.
5. Aortic aneurysm.
6. Pancreatic mass & pseudopancreatic cyst.
6. Retroperitoneal mass.
8. Para aortic lymph nodes.

[C] Causes of unexplained loss of weight in old patient : e.g. cancer caecum.

Investigations:

1. Blood picture : may reveal microcytic anemia.

2. Barium meal :

- 75% accuracy.
- Should include views taken in Trendelenburg's position to detect growth situated in fundus & cardia.
- It may show :

(a) If it is malignant ulcer : it will appear as persistent irregular filling defect.
(b) An ulcer niche outside the ulcer bearing area.
(c) In linitis plastica : Marked narrowing of the lumen of the stomach but the flow of barium is not interrupted.
(d) Malignant pyloric obstruction.

3. Endoscopy : Multiple biopsies should be taken. The most important investigation.

4. Abdominal ultrasonography : For liver secondaries.

5. CT scan : For the detection of lymph node involvement.

6. Estimation of CEA antigen.

Treatment:

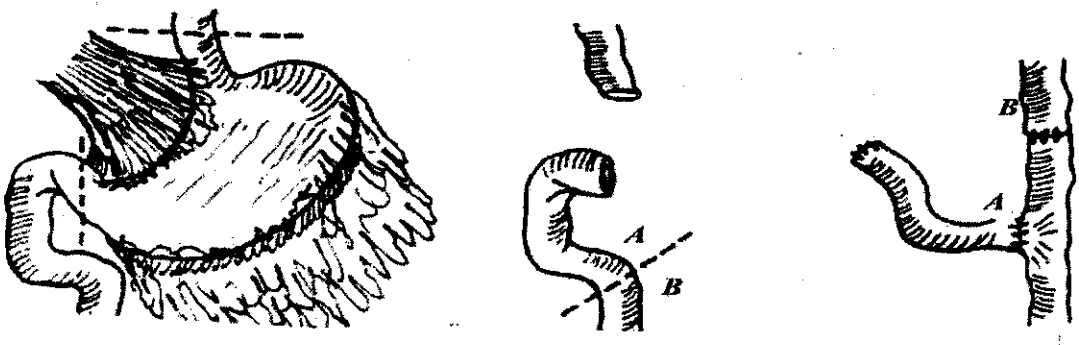
[A] Operable cases :

- **The aim of treatment is cure.**
- **Methods :** Surgery with radical removal of :

1. The tumor with safety margin of at least 5 cm above it and 1.5 cm of the duodenum below it.
2. Both omenta.
3. All the draining lymph nodes.

- **According to the site of the neoplasm, the extent of resection will be determined :**

Site of the tumor	Extent of resection	Restoration of gut continuity
Tumors of the **lower** third of the stomach.	A lower radical partial gastrectomy.	Anastomosis of the remaining upper stomach to the jejunum.
Tumors of the **middle** third of the stomach.	Radical total gastrectomy.	The esophagus is anastomosed to a **Roux-en-Y** loop of jejunum.
Tumors of the **upper** third of the stomach.	Esophagogastrectomy through a thoracoabdominal incision.	The esophagus is anastomosed to a **Roux-en-Y** loop of jejunum.



Roux-en-Y

[B] Inoperable cases :

I- Palliative procedures :

1. Palliative gastrectomy.
2. For irremovable tumors in the pyloric region an anterior gastrojejunostomy is performed.
3. For irresectable lesions in the upper stomach an esophagojejunostomy can be performed.

II- Radiotherapy and chemotherapy :

- These are used in the inoperable, non-resectable, incompletely resected or recurrent cases.
- Of little value.

Prognosis :

Depends on :

1- Histological type and differentiation.
2- The depth of penetration of the wall.
3- The presence or absence of lymph node involvement.

- **Only about 40% of patients are found to be operable on exploration and only 5% are alive 5 years after resection.**
- **Because of early detection in Japan the 5-year cure rate is between 80 % and 90%.**

Gastrointestinal stromal tumors (GIST)

- Usually found in the stomach or small intestine but can occur anywhere along the GIT.
- Vague, nonspecific abdominal pain or discomfort is the most common presentations.
- **Surgery** is the definitive therapy (local gastric resection).
- **Imatinib** (tyrosine kinase inhibitor): Adjuvant therapy post complete surgical resection in patients with high-risk tumors.

Acute gastric dilatation

Definition : This is a condition where the stomach loses its tone.

Etiology :

1. Post-operatively especially after pelvic operations, splenectomy and cholecystectomy, but very rarely after gastric surgery.
2. During labor.
3. Fractures.
4. Debilitating or toxemic states.

Clinical features :

- Hiccough.
- Upper abdominal discomfort.
- The condition should be diagnosed at this stage, if not, there will be :

1. Vomiting : Dark foul smelling fluid.
2. A succussion splash.
3. Upper abdominal pain.
4. Circulatory collapse with death rapidly.

Treatment :

- Is essentially prophylactic by pre and postoperative gastric decompression.
- The occurrence of the condition indicates mismanagement.

Established cases :

- Immediate passage of a Ryle's tube with continuous aspiration.
- Attention to the chest.
- Correction of hypovolemic shock.
- No surgical treatment.

Bariatric surgery

$$BMI = \frac{Weight(kg)}{Height^2(m^2)}$$

- Bariatric surgery is weight reduction surgery
- It can be open or laparoscopic

Complications of obesity

The higher grades of obesity, the worse are the complication

BMI	Classification
18-25	Ideal weight
25–30	overweight
30–35	mild obesity
35–40	moderate obesity
≥ 40.0	morbid obesity

1. **Physical health complications**
   1. Diabetes mellitus & hypertension.
   2. Sleep apnea.
   3. Coronary heart disease.
   4. Spine degenerative disease and disc prolapsed.
   5. Osteo-arthrosis.
   6. Thrombo-embolism.
   7. Difficult vascular access.
   8. GERD.
2. **Mental health complications**; depression
3. **Social complications**; difficulties to get certain jobs, with transportation and with closing

- Bariatric surgery commonly causes loss of 55-75% of excess body weight within 2 years and greatly reduce the risk of obesity co-morbidities

Prerequisites for surgery:

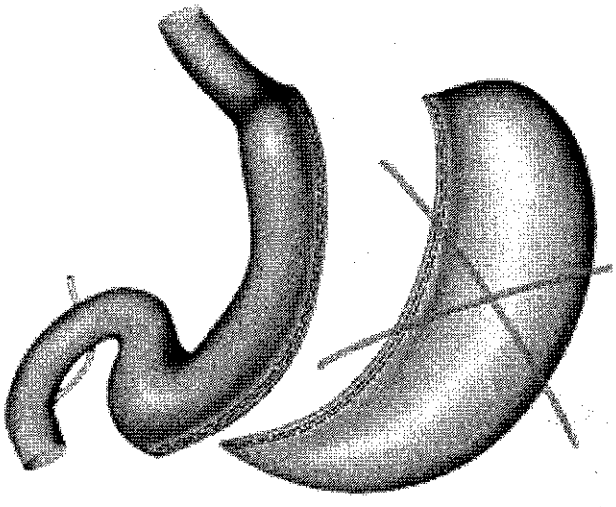
- **BMI > 40** and some cases of BMI 35–40 if co-morbid present (DM & hypertension)
- Failure of conservative measures of weight reduction.
- Age above 18 years.
- Exclusion of other causes of obesity.
- Patient is well informed.

It induces weight reduction through

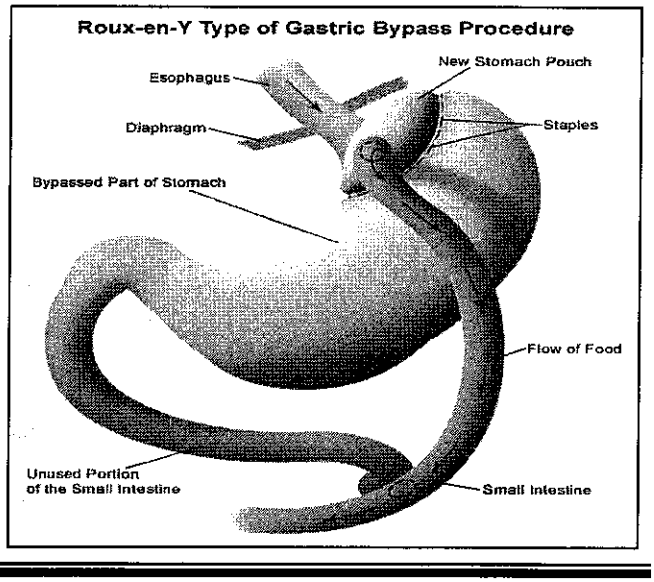
- **Restricting food intake**
- **Inducing malabsorption** ; a part of small intestine is bypassed
  - ✓ Commonly bariatric operations combine malabsorption with restriction of food intake.
  - ✓ The patient should receive life-long vitamins and minerals supplements

Bariatric Surgical Procedures;

- **Sleeve gastrectomy**



- **Roux-en-Y gastric bypass**



Biliary System

Congenital abnormalities of the gall bladder & the bile ducts

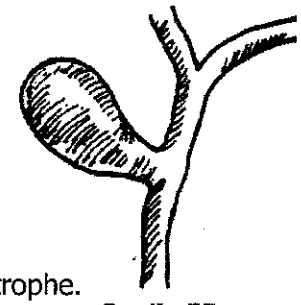
Congenital anomalies of the biliary tract are found in 10 percents of autopsies.

Anomalies of the gall bladder :

1. Absence of the gall bladder : Is rare occurring in 0.03% of autopsy cases.
2. The phrygian cap : Is kinking of the fundus of the gall bladder and is present in 2 to 6 percent of cholecystographies. It is of no clinical significance.
3. Double gall bladder : With two separate cavities and cystic ducts occurs in 1 in 4000.
4. Floating gall bladder : The organ hangs on a mesentery which makes it liable to torsion.
5. Left sided gall bladder : Is extremely rare.
6. Intrahepatic gall bladder : Can occur and is associated with an increased incidence of gall stones.

Anomalies of extra hepatic ducts :

1. Biliary atresia.
2. Choledochal cyst.
3. Hepatic duct anomalies :
(a) Accessory hepatic duct may enter into the cystic duct or into the neck of gall bladder.
(b) The right, left or even both hepatic ducts enter the gall bladder.
Ligation of these ducts during cholecystectomy could result in a surgical catastrophe.


Sessile GB

Cystic duct anomalies :

1. Absence of the cystic duct : Injury of the common bile duct is liable to occur when cholecystectomy is performed in a patient with this abnormality.
2. Low insertion of cystic duct : The cystic duct opens into the common bile duct near the ampulla. This anomaly is common and it may lead to ligation of the common bile duct during a cholecystectomy operation.
3. The cystic duct may enter the right hepatic duct and the latter may be mistaken for the cystic duct and ligated.

Anomalies of the hepatic and cystic arteries :

1. Accessory cystic artery : (the commonest anomaly) may be torn if it is not identified.
2. A large accessory left hepatic artery : may arise form the left gastric artery in about 5% of people.
3. The right hepatic artery : may arise from the superior mesenteric artery.
4. The right hepatic artery : may pass in front of or behind the common hepatic duct or common bile duct and may be mistaken for the cystic artery and, therefore, ligated.

Congenital biliary atresia

Pathology : Types of biliary atresia :
1. Correctable type (10%) : There is a patent portion of the extrahepatic duct which communicates with the intrahepatic ducts.
2. Non correctable type (90%) : The extrahepatic portion of the biliary tract is occluded.

Clinical Picture :

Obstructive Jaundice present since birth, but may not be marked until after the first few weeks.

Differential diagnosis :

This includes physiological jaundice, hemolytic diseases, neonatal hepatitis, α_1-antitrypsin deficiency, the inspissated bile syndrome, infection with different viruses or metabolic defects.

Treatment :

1. Correctable type : A Roux-en-Y jejunal loop is anastomosed to patent portion of the extrahepatic biliary tract.

2. Non correctable type :

Portoenterostomy **(Kasai operation)** in which minute biliary channels present at porta hepatis are anastomosed to a loop of jejunum. If portoenterostomy fails, liver transplantation becomes the treatment of choice.

Choledochal cyst

Rare congenital anomaly in which there is fusiform dilatation of the common bile duct.

Clinical features :

- Usually diagnosed in childhood before 10 years of age.
- Obstructive jaundice.
- Right hypochondrial mass are the main clinical presentations.
- Cholangitis may occur.

Treatment :

The cyst should be excised and choledochojejunostomy is performed.

Radiological investigations of the biliary system

[1] Plain X-ray :

- Detect gall stones in only 10% of cases.
- Gall stones are usually multiple faceted, signet ring and are anterior to the spine in lateral view.

[2] Abdominal ultrasound :

1. Detection of gall bladder stones in a 98% of cases. Less accurate in CBD stones.
2. Detection of the thickness of the wall of the gall bladder.
3. Visualization of dilatation of intra- & extrahepatic biliary radicles.
4. Detection of masses in the porta hepatis, or head of pancreas.

[3] Oral cholecystography :

Now it has been nearly replaced by ultrasonography.

Method : The patient fasts for 12 hours before the examination and swallows 6 tablets of **telepaque** (tetraiodophenolphthalein).

- The contrast material is absorbed, secreted by the liver in the bile and passes via the cystic duct to be concentrated in the gall bladder.
- **In normal persons the gall bladder is well opacified.**
- The patient is then given a fatty meal and another film is taken to assess the contractile power of the gall bladder which should shrink to about one third or its size.
- **Radiolucent stones will appear as filling defects.**
- **Failure of the gall bladder to opacify may be due to :**

1. Vomiting or failure of absorption.
2. Inability of the liver to secrete the dye, e.g. in jaundice.
3. Presence of cystic duct obstruction.
4. Severely inflamed gall bladder with loss of its concentrating power.

Contraindications : If bilirubin is > 3mg%.

[4] Intravenous cholangiography :

- **Biligrafin dye** is injected intravenously (after testing for allergy); it is secreted by the liver and visualizes the biliary tree and the gall bladder.
- Has been replaced now by **ERCP** or **PTC.**

[5] Endoscopic retrograde cholangiopancreatography (ERCP) :

Method :

- An endoscope is passed inspecting esophagus, stomach and duodenum.
- The duodenal papilla is localized and a cannula is passed in it.
- A contrast material is injected to visualize the common bile and the pancreatic duct by radiography.

Indications :

1. Diagnosis of lesions involving the lower end of CBD as carcinoma of head of pancreas or ampulla of Vater. A biopsy can be taken from an ampullary lesion.
2. Detection of calculi in the CBD after cholecystectomy.
3. Detection of operative injuries of the biliary system
4. Visualization of the pancreatic duct in patients with chronic pancreatitis or pseudopancreatic cyst.
5. Therapeutic procedures e.g. sphincterotomy, insertion of a stent.

[6] Percutaneous transhepatic cholangiography (PTC) :

- **Method :** Under local anesthesia, a thin cannula (Chiba needle) is introduced through the 8th intercostal space into the liver and continuous suction by a syringe is applied until bile is aspirated and the dye is then injected the dye will visualize the intrahepatic biliary ducts and then it will flow to the duct from above downwards.

- **Indication :** Diagnosis of obstruction of the bile duct.

- **Precaution :** Prothrombin concentration should be > 60%.

- **Complications of PTC :**

1. Bleeding if there is a disturbance of the coagulation mechanism.
2. Biliary peritonitis.
3. Cholangitis.

[7] Operative cholangiography : During cholecystectomy to detect any stones in the CBD.

[8] T-tube cholangiography.

[9] Radioisotope scanning.

[10] Magnetic resonance cholangiopancreatography (MRCP).

Female predominance is not present with pigment stone or after the age of 60.
Crohn's disease, small bowel resection or bypass procedure are predisposing to Gallstones.
D.M via autonomic neuropathy of the ileum may affect the enterohapatic circulation and predisposing to Gall stones.
Prophylactic cholycystectomy for silent stones are rarely indicated.
Gallstones ileus is a **rare** complication.
In Calcular Cholecystitis, HIDA scan is not commonly performed.
In Cholecystectomy, the primary hemorrhage may be due to injury of the cystic A, RT hepatic A, or from The G.B fossa.
Antibiotic used in acute Cholecystitis **must** be effective against gram -ve bacilli.
It is a trend now to treat acute Cholecystitis by early surgery.

Gall stones

Incidence :

- More common in westernized countries.
- There is also familial incidence of the disease.
- A female to male ratio of 3 : 1 is reported in cholesterol stones.
- The incidence of gall stones increases with age.

Female predominance is **not** present with pigment stone or after the age of 60.

Types and composition : Gall stones are now classified into two categories :

1. **Cholesterol stones.**
2. **Pigment stones;** black and brown.

	Mixed cholesterol stones	Pure cholesterol stones	Black pigment stones	Brown pigment stones
Number	Multiple	Single	Multiple	Multiple
Color	Yellowish	Yellowish	Tarry black	Brown
Shape	Faceted	Oval or rounded	With multiple specules	Laminated
Size	0.5-2.5cm	More than 2.5cm	Less than 2.5cm	Less than 2.5cm
Structure	Cholesterol (60%) + calcium bilirubinate, calcium palmitate	100% Cholesterol	Calcium bilirubinate	Calcium bilirubinate (the major part) + Ca palmitate & Cholesterol
Incidence	90% of all calculi		Found in patients with hemolytic anemia or cirrhosis	They may be formed in the GB or in the bile ducts
X-ray	Radio-opaque in 20% of cases	Radiolucent	Radio-opaque in 50% of cases	Radio-opaque

Etiology of cholesterol gall stones :

[A] Disturbed bile salts cholesterol ratio :

- A certain ratio (25: 1) between bile salts and phospholipids on one hand and cholesterol on the other hand has to be maintained to keep cholesterol in solution.
- Any lowering of this ratio can lead to supersaturated bile (lithogenic bile) with consequent cholesterol precipitation.

I- Reduced bile salt in :

1. Malabsorption of bile salts in the terminal ileum. **Crohn's disease - Small bowel resection**
2. Diminished hepatic synthesis in liver disease.
3. Estrogen reduces the concentration of bile salts in bile.

II- Increased cholesterol synthesis : e.g. in obesity, high dietary fat.

[B] Stasis : This may occur in the following conditions :

1. Pregnancy & contraceptive pills : progesterone causes relaxation and impaired emptying of the GB.
2. Following truncal vagotomy.
3. Diabetes mellitus. *Autonomic neuropathy of the ileum may affect the enterohapatic circulation*
4. Obesity.
5. Long term parenteral nutrition.

[C] Infection.

Etiology of pigment stones :

1. Hemolytic anemia.

2. Hepatic cirrhosis: Most probably due to decreased secretion of bile acids by the cirrhotic liver leading to diminished solubility of bilirubin.

3. Infection : Infection by some strains of E. coli leads to formation of insoluble bilirubin which precipitates as calcium bilirubinate.

Clinical picture of gall stones :

[1] Silent gall stones : Accidentally detected by **X-ray** or **U.S.**

[2] Recurrent attacks of biliary colic :

- **Severe colicky upper abdominal pain** which may be referred to the back of the right chest or right shoulder.
- **Usually precipitated by the intake of a fatty meal.**
- **Clinical examination may reveal :**

1. Tenderness in the right hypochondrium.
2. **Murphy's sign :** May be positive (the gall bladder area is palpated while the patient is asked to take a deep breath; the patient will catch her breath).

[3] Biliary symptoms : A common complaint is the occurrence of fatty dyspepsia with flatulence and excessive eructation.

[4] Reflex symptoms : Gall stones may cause reflex retrosternal pain which is usually diagnosed as anginal pain. Actual ECG changes may occur.

[5] Complications :

[A] Migration and obstruction :

1. Obstruction of the cystic duct or Hartmann's pouch : This will lead to either mucocele of the gall bladder or empyema.
2. Obstruction of the CBD : Will lead to calcular obstructive jaundice.
3. Obstruction of the ampulla of Vater : May precipitate an attack of acute pancreatitis.
4. Obstruction of the terminal ileum (Gall stone ileus) : **Rare**

- Occurs secondary to a fistulous communication between the gall bladder and the duodenum.
- A big stone passes through this opening and usually gets impacted in the terminal ileum about two feet from the ileocecal valve.
- Clinical diagnosis is usually difficult as the patient is often elderly and gives a long history of biliary problems. The symptoms of intestinal obstruction are usually misinterpreted as being due to biliary problems.

[B] Infection from stasis :

1. Acute calcular cholecystitis.
2. Chronic calcular cholecystitis.
3. Cholangitis (discuss in short).
4. Acute Pancreatitis.

[C] Carcinoma of the gall bladder.

[N.B 1] Wilkie's triad :

1. Chronic peptic ulcer.
2. Chronic appendicitis.
3. Chronic calcular cholecystitis.

[N.B 2] Saint's triad :

1. Hiatus hernia.
2. Diverticular disease of the colon.
3. Chronic calcular cholecystitis.

D.D. : Other causes of dyspepsia.

Investigations of gall stones : (see general)

1. Routine laboratory tests : Liver function tests.

2. Abdominal ultrasound (the investigation of choice).

3. Plain X-ray.

4. Oral cholecystography :

- This is rarely performed now.
- It may be indicated to assess the contractility of the gall bladder after a fatty meal to diagnose patients with biliary dyskinesia.

Treatment of gall stones

Silent gall stones

Prophylactic cholecystectomy is rarely indicated as they remain asymptomatic in 70 % of people over 20 years. It may be advised in elderly diabetics (if acute cholecystitis developed, it will be serious).it is indicated in gall bladder wall calcification (porcelain gall bladder) which is precancerous.

Patients with symptomatic gall stones

The standard is cholecystectomy, either open (conventional) or laparoscopic.

N.B.: ESWL & medical dissolution of gallstones have been dropped out of use for there many side effects.

Laparoscopic cholecystectomy (LC) :

The idea of the procedure is :

- To induce a pneumoperitoneum using CO_2 gas.
- Then through 4 small ports, a special camera and fiberoptic scope are introduced and a magnified picture of the internal organs is visualized on a television screen.
- By using special graspers and instruments, the surgeon can perform cholecystectomy following nearly the same steps as in open surgery.

Advantages :

1. **Post-operative pain** is less.
2. **Post-operative stay** in the hospital is short.
3. **Post-operative convalescence** is more smooth allowing early return to work.
4. **Better cosmetic result.**

Contraindications to LC :

1. **Pregnancy :** there is no room to induce pneumoperitoneum and CO2 gas may be toxic to the fetus.
2. **Bleeding tendency.**
3. **Carcinoma of the gall bladder.**
4. **Compromise of the cardiovascular or respiratory functions.**

Difficult cases :

1. **Marked obesity** as it is difficult to introduce the ports.
2. **Liver cirrhosis.** Major bleeding may occur from the bed of the gall bladder.
3. **Empyema of the gall bladder.**
4. **Previous upper abdominal surgery is a relative contraindication.**

N.B.: A wise surgeon should convert the procedure to open surgery without any hesitation, when indicated.

Acute cholecystitis

In 98% it is secondary to obstruction of the cystic duct or Hartmann's pouch by a stone.

Acute calcular cholecystitis

Pathology :

1. Obstruction of the cystic duct or neck of gall bladder results in **chemical irritation** of the gall bladder wall by the bile salts.
2. If the bile remains sterile, the gall bladder becomes distended by mucous and a **mucocele of the gall bladder** develops.
3. More often, secondary infection by gram negative bacilli occurs and **empyema of the gall bladder** develops.
4. The gall bladder is distended, its wall is thickened, and its serosal lining loses its normal bluish luster and is covered by adhesions.
5. The greater omentum, the duodenum and colon become adherent to the gall bladder to localize the inflammation.
6. A special form called **acute emphysematous cholecystitis** may occur in diabetic patients due to infection with anaerobic organisms, e.g. clostridia. The infection is highly virulent & is accompanied by gas formation and leads to early gangrene of the gall bladder.

Consequences :

1. In most cases, with treatment, the stone dislodges and the obstruction is relieved with **gradual resolution** of inflammation.
2. Less commonly the inflammation is more sever with progressive distention of the gall bladder. Thrombosis of the blood vessels & perforation of the gall bladder may occur followed by either localized or less commonly generalized peritonitis (0.5%).
3. Recurrent attacks lead to **chronic calcular cholecystitis.**
4. Secondary infection by gram negative bacilli occurs and **empyema of the gall bladder** develops.

A special form called **acute emphysematous cholecystitis** may occur in diabetic patients due to infection with anaerobic organisms e.g. clostridia. The infection is highly virulent & is accompanied by gas formation and leads to early gangrene of the gall bladder.

Clinical picture :

Symptoms :

1. Pain :

- Initially it is diffuse colicky upper abdominal pain.
- With inflammation of the serosal lining, the pain becomes dull aching, persistent and localizes in the right hypochondrial region.
- The pain may be referred to the right shoulder due to irritation of the under surface of the diaphragm supplied by sensory fibers of the phrenic nerve.

2. There is usually nausea, or sometimes vomiting.

Examination :

- **General :** Usually there is pyrexia and tachycardia.
- **Abdominal examination :**

1. Tenderness, rebound tenderness and muscle guarding in the right hypochondrial area.
2. G.B. mass : It is difficult to palpate the distended gall bladder due to the overlying tenderness and rigidity.
3. Boas's sign : Sometimes, an area of hyperesthesia may be elicited between the 9th to the 11th ribs posteriorly on the right side.
4. Mirrizi syndrome : Rarely, the distended Hartmann's pouch containing the stone may compress the bile duct leading to jaundice.

Differential diagnosis :

1. Leaking duodenal ulcer. 2. Acute pancreatitis.
3.Subhepatic appendicitis. 4. Right pyelonephritis. 5. Amoebic hepatitis.

Investigations :

[A] Laboratory :

1. Blood picture : polymorphonuclear leucocytosis may be present.
2. Liver function tests : Are usually normal.

[B] Radiological :

1. Abdominal ultrasound : the most appropriate investigation. It will reveal the presence of gall stones, distended gall bladder, thickened wall and it can also reveal microabscesses and serosal edema due to spreading inflammation.
2. HIDA Scan: ***Not Commonly Performed***
^{99m}Tc is administered IV. It is secreted by the liver, and is then excreted along the biliary pathway. Normally it is concentrated in the gall bladder. If the common bile duct is visualized while the gall bladder is not seen, this is diagnostic of acute cholecystitis.

Treatment : There are two options available :

[1] Early cholecystectomy :(within 3 days of onset of the attack)

- **Early cholecystectomy is now recommended so long as :**

1. The diagnosis is confident. 2. Patient is fit. 3. The surgeon is competent.

- **Advantages :**

1. Early surgery (within 2-3 days) is not unduly difficult.
2. Avoids the complications of acute cholecystitis which may arise during conservative treatment.
3. One hospital admission with early return to work.

[2] Conservative treatment followed by surgery after 6 weeks :

- The other policy is to treat the patient conservatively as following :

1. **Nil by mouth, and IV fluids.**
2. **Antibiotics.** ***Must be effective against gram –ve bacilli***
3. **Analgesics** as pethidine or NSAIDs.
4. **Follow-up** of the pulse, temperature, degree of pain and the area of tenderness and rigidity.

- Most of the patients improve and the attack subsides.
- The patient is sent home & is advised to come back after 6 weeks to have cholecystectomy operation.
- If the patient deteriorates, surgery should be performed and either cholecystectomy or cholecystostomy is performed according to circumstances.

- **Advantages of this policy :**

1. Most of the patients will improve by conservative treatment.
2. Surgery in the acute stage is difficult as the tissues are friable, edematous and there are adhesions making identification of important structures difficult.

[3] Emergency cholecystectomy or cholecystostomy :

Indications :

1. Doubtful diagnosis (appendicitis or duodenal ulcer).
2. Failure of medical treatment after 2 days with increasing pulse, pain, temperature & TLC.
3. Complications e.g. mass and perforation.
4. Diabetes mellitus or typhoid.

N.B: in unfit Patients: percutaneous drainage of the gall bladder can be done.

Why acute appendicitis is treated by surgery while acute cholecystitis may be treated conservatively ?
1. The organisms causing acute appendicitis are much more virulent.
2. The appendix has only the appendicular artery for its supply while the gall bladder has numerous vascular connections to the undersurface of the liver in addition to the cystic artery.

Acute non-calcular cholecystitis

- This is a rare and serious condition.

Etiology : The actual cause and pathogenesis of the condition is unclear.

- **Change in the composition of bile** or **ischemia of the gall bladder** may be responsible.

1. It may occur in patients suffering from major burns or major trauma.
2. It may complicate certain infections such as brucellosis and typhoid fever.
3. Prolonged total parenteral nutrition.

Clinical Picture : It is similar to that of acute calcular cholecystitis, but usually the diagnosis is delayed because it is not suspected.

Investigation : Ultrasound examination is diagnostic.

Treatment : Is by urgent cholecystectomy.

Strictures of the biliary tract

- Etiology and types :

[1] Congenital biliary atresia.

[2] Traumatic strictures : Usually follows cholecystectomy or less commonly choledocholithotomy.

- **It may be due to :**

1. Complete ligation of the common hepatic or common bile duct.
2. Narrowing of the duct due its inclusion in a ligature.
3. Ischemia of the duct.
4. Devitalization of the duct due to rough dissection.

- These patients present by either biliary fistula or jaundice following cholecystectomy.
- ERCP and PTC will localize the site of the stricture which is usually at the hepatic duct.
- **Treatment :** The remaining stump of the common hepatic duct if present or the left hepatic duct is anastomosed to a Roux-en-Y loop of jejunum.

[3] Inflammatory stricture (primary sclerosing cholangitis) :

- **Etiology :** Unknown.
- **Pathology :** The intra-and extrahepatic ducts are involved by multiple strictures separated by normal or dilated segments.
- **Clinical picture :** The patient presents by obstructive jaundice and if the condition is untreated, it will eventually lead to liver failure.
- **Treatment :** These patients are candidates for liver transplantation.

[4] Neoplastic stricture : Cholangiocarcinoma :

- **Incidence :** Elderly males.
- **Predisposing Factors :** Calcular disease, ulcerative colitis, primary sclerosing cholangitis, choledochal cyst and parasitic infestation (clonorchiasis).
- **Clinical Picture :**

1. They manifest by obstructive jaundice in 90% of patients.
2. Pain, cholangitis and anorexia.
3. If the tumor is below the insertion of the cystic duct into the CBD, the gall bladder will be distended.
4. If the cholangiocarcinoma is at the porta hepatis **(Klatskin's tumor),** the gall bladder will be collapsed **(an exception to Courvoisier's law).**

- **Investigations :** ultrasound & PTC.
- **Treatment :**

1. Operable lesions at the lower end of CBD are treated by pancreatico - duodenectomy **(Whipple's operation).**

2. Inoperable cases are treated by cholecystojejunostomy.

3. Lesions at the hilum of the liver are rarely operable. Treatment is by anastomosis of any dilated biliary segment above the lesion to a loop of jejunum, or if this is not possible by some form of biliary drainage as percutaneous transhepatic biliary drainage or endoscopic stenting.

Carcinoma of the gall bladder

Incidence :

This lesion is more common in elderly females. In over 90% of cases gall stones are present.

Pathology :

- Microscopic picture is that of squamous cell carcinoma, adenocarcinoma or a mixture of the two.
- Spread is by direct invasion of the liver and porta hepatis, by lymphatics to the hilar lymph nodes or venous spread to the liver.

Clinical picture :

1. The tumor is usually diagnosed only after examination of the gall bladder removed by cholecystectomy or by pathological examination.
2. Some patients present by acute cholecystitis as the tumor obstructs the cystic duct.
3. Obstructive jaundice due to obstruction of the bile duct by the tumor or by metastatic lymph nodes in the porta hepatis.
4. Mass in the right hypochondrium.

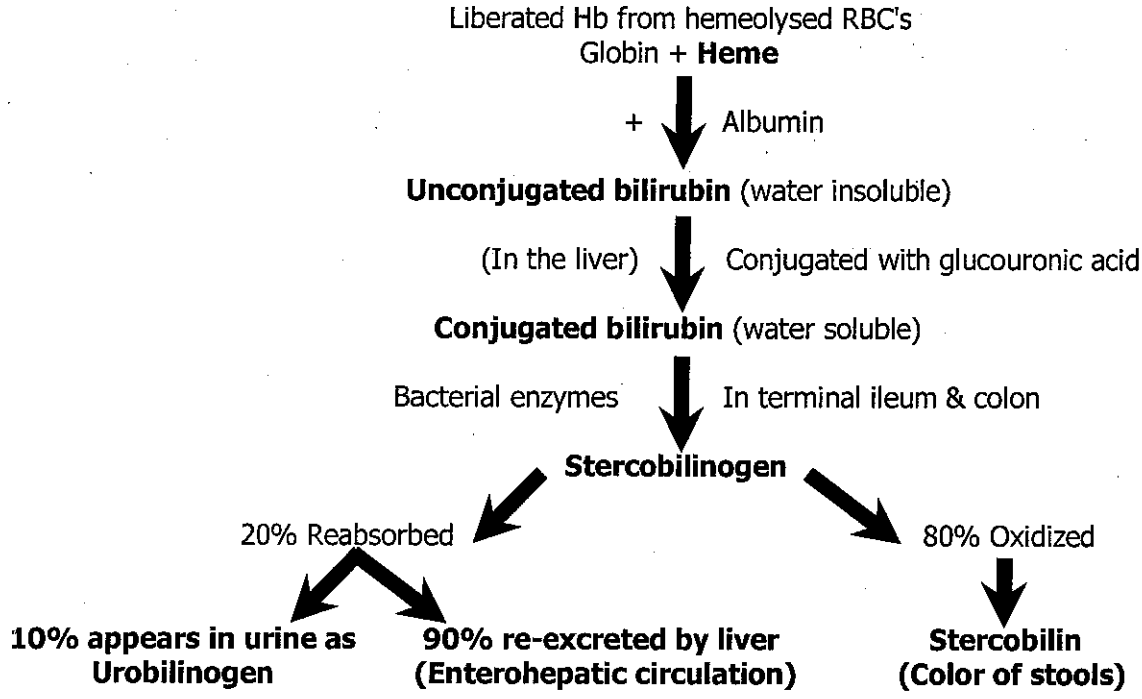
Treatment :

- If the diagnosis is suspected during surgery, the treatment is cholecystectomy plus excision of a wedge of the underlying liver and if possible clearance of any lymph nodes overlying the bile duct.
- Usually the lesion is fairly advanced and inoperable. If there is jaundice, internal stenting may relieve it. The prognosis for this lesion is very poor.

Jaundice

Jaundice is yellowish coloration of the body tissues and fluids (except the brain, CSF, tears, saliva and milk) which result from accumulation of bilirubin in blood.

Bilirubin metabolism


Liberated Hb from hemeolysed RBC's
Globin + Heme
+ Albumin
Unconjugated bilirubin (water insoluble)
(In the liver) Conjugated with glucouronic acid
Conjugated bilirubin (water soluble)
Bacterial enzymes In terminal ileum & colon
Stercobilinogen
20% Reabsorbed
80% Oxidized
10% appears in urine as Urobilinogen
90% re-excreted by liver (Enterohepatic circulation)
Stercobilin (Color of stools)

Obstructive Jaundice

Causes of obstructive jaundice include :

[A] Intra hepatic cholestasis (medical) :

1. Viral hepatitis.
2. 2ry biliary cirrhosis.
3. Drugs : • Hypersensitivity reaction e.g. choloropromazine. • Dose dependent e.g. testosterone.
4. Dubin-Johnson and Rotor syndromes.

[B] Extra hepatic cholestasis (surgical) :

From inside	From wall	From outside
1. Stone. **2. Blood.** **3. Parasite** e.g. Ascaris, hydatid or fasciola.	**1. Atresia.** **2. Traumatic :** stricture is usually iatrogenic following cholecystectomy, choledocholithotomy or instrumental. **3. Papillary stenosis.** **4. Sclerosing cholangitis.** **5. Tumors of the duct** e.g. cholangiocarcinoma.	1. Choledochal cyst. 2. Duodenal diverticulum. 3. Pancreatic tumor or pancreatitis. 4. Metastatic L.N. in the porta hepatis.

Calcular Obstructive Jaundice (Choledocholithiasis)	Malignant Obstructive Jaundice
Pathology	
Cause : Obstruction of bile ducts by a stone. **Site of obstruction :** • Usually, in common bile duct. • Rarely, in common hepatic duct. • Very rarely, a stone in Hartmann's pouch can enlarge and compress CBD **(Mirrizi Syndrome).** **Origin of stone :** In most cases, a stone in the common bile duct (CBD) is originally formed in the gall bladder, and has slipped through the cystic duct into the CBD. • 15% of patients with calcular cholecystitis have stones in the CBD. • Primary stones in the CBD are rare and occur when there is prolonged stasis and infection.	**Causes :** 1. Carcinoma of head of pancreas. 2. Carcinoma of duodenal papilla. 3. Carcinoma of ampulla of Vater. 4. Carcinoma of bile ducts. 5. Liver tumors (primary or secondary). 6. Malignant lymph nodes in porta hepatis. 7. Adenocarcinoma of gall bladder infiltrating bile ducts.

Sequelae of CBD stones

1. No effect : if the stone remains floating in CBD without obstruction.
2. Passage to the duodenum : stone < 3mm, can pass spontaneously through the sphincter of Oddi. During its passage it may produce acute pancreatitis.
3. Obstruction of CBD :
• Obstructive jaundice.
• Dilatation of the bile duct and intrahepatic biliary radicles.
• In the presence of prolonged obstruction and high pressure in the bile ducts, bile secretion by the liver stops. The bile ducts become full of mucous (white bile).
• **Complications :**
(a) Bleeding tendency. (b) Cholangitis. (c) Septicaemia and hepatorenal failure
(d) Acute pancreatitis : if stone impacts in lower part at common channel of pancreatic duct & CBD.
(e) Biliary cirrhosis is uncommon. It occurs in prolonged cases where obstruction is intermittent or incomplete.

Clinical picture

I- Age : Middle age (forty, fifty).	**I- Age :** Usually old age.
II- Sex : Usually female (fatty, filthy, fertile).	**II- Sex :** Usually male.

III- General Clinical Picture of obstructive Jaundice :

1. Jaundice : bilirubin metabolism continues until conjugated (direct) bilirubin is formed by liver and then retained in bile ducts where it is absorbed into blood.

2. Dark urine (like tea) : Conjugated bilirubin, which is now abnormally present in blood, is excreted in urine darkening its color.

3. Pale (clay colored) stool : As bilirubin fails to reach intestine (due to obstruction). No sterchobilin is formed and stools are

Also there is decreased formation of urobilin (detected chemically).

4. Bile salts :

- Accumulate in the blood causing :

Itching, drowsiness, headache, irritability, bradycardia & hypotension.

- Fail to reach intestine, resulting in :

i- Defective fat absorption : bulky stools & impaired vitamin absorption (fat soluble) leading to prothrombin deficiency & bleeding tendency.

ii- Offensive stools.

iii- Anorexia, abdominal distension and constipation.

- Appear in urine (detected chemically).

IV- Specific Symptoms :

1. Pain : The patient may complain of recurrent attacks of severe dull aching pain in the right hypochondrial and epigastric regions which may be referred to the right scapular region or even to the back. During the attacks there may be nausea, vomiting. **2. Charcot's triad :** due to superadded cholangitis, the patient gives a history of recurrent attacks of pain, jaundice, fever and rigors. **N.B. : Reynold's pentad :** Charcot's triad with mental confusion and hypotension (shock).	**Usually painless.** There may be epigastric pain radiating to back.
V- Course : Intermittent.	**V- Course :** Progressive. (Rarely, jaundice may regress if central degeneration occurs in the tumor with recanalization).

VI- Abdominal signs :

1. Courvoisier's law. 2. Enlarged tender liver.	1. Courvoisier's law. 2. Enlarged tender liver. 3. Signs of malignancy : e.g. Abd masses, Ascitis.

N.B.: Clinical picture of choledocholithiasis :

1. May be silent.	2. Pain (see before).
3. Calcular obstructive jaundice (see before).	4. Charcot's triad.
5. Acute pancreatitis.	6. Picture of associated gall stones.

Differential Diagnosis :

1. Obstructive from other types of jaundice.
2. Calcular from malignant.

Investigations (6 Problems)

[1] To prove jaundice :

Estimate serum Bilirubin :

1. Normal to 1mg.
2. 1-3 subclinical jaundice.
3. Over 3 clinical Jaundice.
4. In calcular obstructive jaundice it rarely exceeds 10mg.

[2] To prove that it is obstructive :

1. Stool :

- Clay colored, bulk, offensive.
- Sterchobilin is decreased (detected chemically).

2. Urine :

- Dark (like tea)
- Urobilin is decreased (detected chemically).
- Bilirubin & bile salts are detected chemically (normally absent).

3. Raised Bilirubin is mainly direct.

4. Alkaline Phosphatase: If other causes of raised alkaline phosphatase are excluded, this enzyme is the most sensitive indicator of biliary tract obstruction.

5. Gamma glutamyl transferase & 5-nucleotidase : Are both maximally elevated in obstructive jaundice.

[3] To exclude other types of jaundice :

Blood examination to exclude hemolytic jaundice.

[4] To assess its effect on coagulation :

(Impaired clotting contraindicates surgery)

- Prothrombin time and concentration are normal in hemolytic jaundice.
- There is prolongation of prothrombin time and diminished prothrombin concentration in both hepatocellular and obstructive jaundice.
- To differentiate both conditions, a course of IV vitamin K is prescribed for few days. In obstructive jaundice the prothrombin parameters will be improved but not in hepatocellular jaundice.

[5] To assess its effect on liver function :

Liver function tests : SGOT - SGPT - albumin.

[6] To prove cause of obstructive jaundice: (stone or malignancy)

1. Abdominal ultrasound : The first investigation to be done. It provides the following information :

- Dilatation of the intrahepatic biliary radicles in patients with obstructive jaundice.
- The diameter of the CBD (Normal - up to 7 mm) is increased. Presence of chronically inflamed gall bladder with calculi points to but is not conclusive evidence that obstruction is due to calculi.
- Presence of stones in the CBD in a good number of cases.
- A mass in the head of the pancreas may be detected.

2. Abdominal CT : Is valuable in demonstrating lesions of the pancreas or metastatic lymph nodes.

3. Ba meal : May show :

- Wide distorted "C" curve of duodenum (pad sign) = Carcinoma of head of pancreas.

4. ERCP : Can detect lesions of the ampulla and a biopsy can be taken. Both the CBD and pancreatic ducts will be visualized. A stone will appear as a filling defect in the CBD. ERCP should not be done during an attack of cholangitis unless a therapeutic procedure is planned because the rise of pressure during injection of the contrast material may lead to severe systemic sepsis.

5. PTC : Is done in patients suspected of having a lesion involving the upper end of the common bile duct e.g. postoperative stricture or carcinoma of the hepatic ducts.

6. MRCP (Magnetic Resonance Cholangio-Pancreatography) : Is recently used.

N.B. : Cholecystography is contraindicated in jaundice.

Management

Pre-operative Management : (3 Problems)

[A] Correct clotting dysfunction :
1. Vitamin K parenterally 10 - 40mg/d.
2. Fresh Blood transfusion.

[B] Protect against liver cell failure :
1. High intake of glucose and liver glycogen to build up the store.
2. Lactulose.
3. Liver Tonics.
4. Colonic wash out.
5. Intestinal antiseptic.

[C] Protect against renal failure :
1. Proper hydration.
2. Mannitol I.V.
3. Bile salts orally : To avoid bacterial over growth → endo toxemia & renal failure.
4. Prophylactic antibiotics.

See Below.	See Treatment of Cancer head of pancreas

Treatment of calcular obstructive jaundice

There are two lines of treatment for calcular obstructive jaundice :
[A] Endoscopic extraction of calculi followed by cholecystectomy whether surgical or by laparoscopy.
[B] Conventional cholecystectomy and choledocholithotomy.

I- Endoscopic extraction of bile duct stones :

- If ERCP is available followed by cholecystectomy.

- **Possible procedures :**

1. **Endoscopic sphincterotomy.**
2. **Sphincter balloon dilatation.**

- **After 1 or 2 :**

Stones can be extracted in 85-90% of patients using Dormia basket forceps or balloon catheters.

- **If extraction fails, crushing of the stone can be performed by either :**

1. **Mechanical lithotripsy** by special crushing wire basket.
2. **Intracorporeal shock wave lithotripsy.**
3. **Extracorporeal shock wave lithotripsy (ESWL).**
4. **Laser lithotripsy.**

- **Complications of endoscopic maneuvers to extract calculi :**

1. **Bleeding :** occurs in 2-9% of cases. This may be due to coagulation defects. The prothrombin time should be checked before the procedure and vitamin K administered IV.
2. **Acute cholangitis :** occurs in 1-3% of cases.
3. **Pancreatitis :** occurs in 1-4% of cases.

II- Conventional cholecystectomy & choledocholithotomy :

• The supraduodenal part of CBD is exposed, opened between 2 stay sutures and all calculi are extracted.

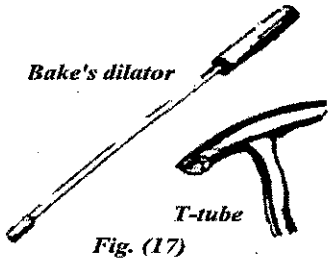


Fig. (17)

• **A special dilator (Backe's)** is passed through the ampulla to check that there is no stenosis.

• **Flushing of the duct with saline.**

• Some surgeons insert a **choledochoscope** to check that there are no retained calculi.

• **A T-tube is inserted in the CBD** which is closed around the tube; the long limb of the tube is brought outside the patient.

• **A T-tube cholangiogram** can be performed after closure of CBD to check absence of filling defects **(Completion T-tube cholangiography).**

• **Cholecystectomy** is then performed.

• **Ten days after surgery a cholangiogram** through the T-tube is performed and if there are no residual stones and there is free flow of the contrast to the duodenum, the tube is removed.

In some cases an additional procedure has to be performed in addition to choledocholithotomy :

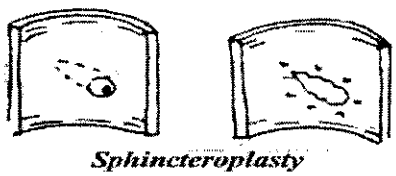
• **Indications :**

1. **Presence of intrahepatic bile duct calculi** which could not be extracted.
2. **Stricture** of the lower end of the CBD.
3. **A stone** impacted in the lower end of CBD.
4. **Dilated duct** with infected bile as here is a possibility of formation of recurrent calculi.

• **Choices include :**

1. **Choledochoduodenostomy :** if CBD > 2 cm in diameter.
2. **Sphincteroplasty :** if CBD < 2 cm in diameter.

Choledecgoduodenostomy


Sphincteroplasty

Indications for exploration of CBD during cholecystectomy :

If ERCP or operative cholangiography is available, the surgeon can ascertain whether calculi are present in the CBD or not. But, if these facilities are not available, exploration of CBD is performed in the following situations :

1. **A stone** is palpable in the CBD.
2. **A dilated CBD** (more than 10mm) with thick fibrous wall.
3. **A dilated cystic duct** especially if there are small gall stones.
4. **Jaundice or history of jaundice.**

Courvoisier's law

Obstructive jaundice due to stones of the CBD is not associated with enlargement of gall bladder, while carcinoma of head of the pancreas is associated with distended gall bladder.

NB: If the Cholangiocarcinoma is at porta hepatis (klatskin's tumour), the G.B will be collapsed

Exceptions of Courvoisier's law are :

1. Enlarged gall bladder in calcular obstructive jaundice in cases of :

(a) Metabolic stone (gall bladder healthy and can dilate).
(b) Stone in common bile duct leading to jaundice and another stone in cystic duct leads to a mucocele of the gall bladder.

2. Collapse of the gall bladder in malignant jaundice :

(a) Association of chronic cholecystitis with carcinoma of the pancreas.
(b) Lymph nodes from malignant pancreas obstruct the common hepatic duct above the cystic duct

Summary of differentiation between calcular & malignant obstructive jaundice

	Calcular	Malignant
Age	Middle age	Old age
Sex	Obese females	Males
Onset	Acute	Gradual
Course	Intermittent	Progressive
Duration	Variable	Short
Pain	Biliary colic	Usually painless
General condition	Good	Cachexia
Jaundice	Moderate	Deep
Fever	May be	Usually absent
GB in Abd. U/S	Fibrosed ± stones	Distended with thin wall
CT scan	Stones in GB	Mass head of pancreas
ERCP	Stone in CBD	Irregular filling defect

	Biliary Colic	Acute Cholecystitis
Pain	Cramp	Steady
Vomiting	May be present	More prominent
Fever	Absent	Present
WBC's	< 10.000	> 10.000
Course	Resolves in 1-2 hours	Usually resolves in 24 - 72 hours

N.B:causes of biliary dyspepsia:

- Chronic calcular cholecystitis
- ***Biliary dyskinesia***:spasm at sphincter of oddi diagnosed by high pressure in CBD (normally 10-18 cm saline) and treated by sphincterotomy
- ***Cholecystosis***:not uncommon group of conditions with chronic inflammatory changes and hyperplasia of all tissues of the gall bladder**.e.g.** Cholesterosis(strawberry gall bladder),cholesterol polyposis of the gall bladder,cholecystitis glandularis proliferns(polyp,adenomyomatosis),diverticulosis of the gall bladder.

Liver

Studies of the liver

I- Liver function tests (LFTs).

II- Imaging of the liver :

1. **Ultrasonography.** 1st choice imaging
2. **CT scan and magnetic resonance imaging (MRI).**
3. **Arteriography :** it is useful in the diagnosis of liver tumors.
4. **Isotope scanning** of the liver.

Segmental anatomy

- Surgically, the liver is divided into **2** almost equal **lobes** by the middle hepatic vein.
- Hepatic veins and their tributaries divide the lobe into sectors, and the sectors into smaller segments.
- There are **four sectors** divided into **eight segments**, and any of them can be excised if its duct and vessels are ligated and divided.
- Each of these segments has its own smaller branch of the bile duct, hepatic artery, and portal vein.
- Segment one, is the caudate lobe that has a special stats as it is supplied by hepatic artery and portal vein branches from both sides & it is drained directly by multiple small veins into IVC.
- Microscopically, the liver is covered by the glisson's capsule.

Liver Trauma

Incidence :

- The liver is the second common solid abdominal organ to be injured.
- Commonly associated with affection of other intra or extra-abdominal organs (especially thoracic).
- Chest stabs as high as the 4th intercostals space can transfix the diaphragm and injure the liver.
- The rate of Iatrogenic liver injuries is increasing.

Pathology :

The types of injury :

1. Small subcapsular hematoma.
2. Small superficial tear or tears.
3. Large subcapsular or intrahepatic hematoma.
4. Large deep tear or tears.
5. Shattered liver parenchyma which may include a whole lobe.
6. Vascular injury: the most difficult to control is that of the main hepatic veins due to difficult access.

Consequences :

- The main danger is bleeding.
- Most liver injuries stop bleeding by the time they are explored. A liver hematoma sometimes communicates with a torn bile duct leading to **"hematobilia".**

Clinical features and diagnosis :

1. History of trauma.

2. The presence of lower rib fracture raises the suspicion of liver affection.

3. Intra peritoneal hemorrhage :

(a) Massive bleeding presents with the picture of hemorrhagic shock.

(b) Minor bleeding discovered by diagnostic peritoneal lavage (DPL), ultrasound or by CT scan, which are done in suspected cases.

4. Abdominal tenderness and rigidity.

5. May also be discovered with **systematic exploration during laparotomy** for penetrating abdominal trauma.

In the unconscious patient it is difficult to assess the abdomen, and for such cases ultrasound, CT or DPL is indicated.

Treatment :

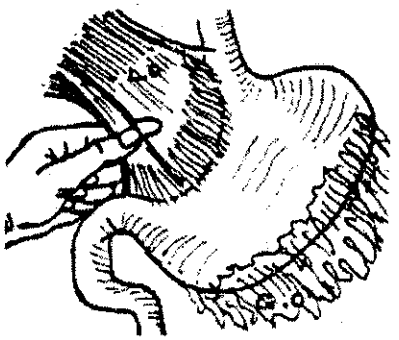
1. Please remember ABCDE.
2. Minor hematomas and small tears: can be conservatively followed.
3. Continuous bleeding and serious liver injuries requiring urgent laparotomy.

The principles of surgical management are :

[A] Adequate exposure by longitudinal incision that can be extended to the chest.

[B] Systematic exploration of the abdomen to assess the liver affection and to detect other intrabdominal injuries.

[C] Dealing with the injuries :



1. Priority is for the arrest of bleeding :

> If bleeding stops by the time of exploration, no treatment is needed, just put a drain.

> Control of liver hemorrhage by a combination of temporarily packing of the bleeding area, and the application of the **Pringle's maneuver** (application of vascular clamp to the free border of lesser omentum, or holding it between two fingers, to occlude the hepatic artery and the portal vein for a period up to 20 minutes). **Then either :**

(a) Visualizing and ligating the bleeding vessels.

(b) Suturing liver tears : should be avoided whenever possible because it is likely to leave a space for accumulation of hematoma that may infect or communicate with intrahepatic bile duct.

- Indications : If control of bleeding vessels is not possible as in deep tears.
- Method : Deep transverse mattress sutures using special needles.

(c) A hematoma is explored to ligate the damaged vessels and ducts, and to excise the dead tissues. It is then left open for drainage.

(d) Firm packing of inaccessible and difficult bleeding areas e.g. with hepatic veins this may be the only method for temporary arrest of bleeding.

2. Excision of the dead tissues.
3. Multiple intraperitoneal drains are always placed to guard against collections of blood and bile.
4. Prophylactic antibiotics are prescribed.

Prognosis : The prognosis is largely depends on associated injuries.
The mortality of liver injury is 15-20%. And 70% if three other major organs are injured.

Infections of the liver

1. Viral hepatitis.
2. Pyogenic liver abscess.
3. Amebic hepatitis and abscess.
4. Hydatid disease of the liver.
5. Hepatic schistosomiasis.

Pyogenic liver abscess

Etiology :

1. Via the portal vein : (In Portal Pyemia) **Streptococci & anaerobes**
(a) Suppurative appendicitis. (b) Diverticulitis.
(c) Infected carcinoma of the colon. (d) Inflamed hemorrhoids.
2. Via the bile duct **Commonest:** In cholangitis. **E. coli**
3. Via the hepatic artery : (In Systemic Pyemia). **Staphylococcus aureus**
4. By direct extension.
5. Cryptogenic.

Clinical picture :

- **General :** The patient is feverish & toxic. **The patient may only present with pyrexia of unknown origin**
- **Local :** Abdominal pain & enlarged tender liver.

Investigations :

1. CBC will show leucocytosis.
2. Abdominal ultrasonography & CT.

Treatment :

1. Drainage with management of the primary cause :
- The abscess is better evacuated through an extraserous approach.
- Broad spectrum antibiotics.

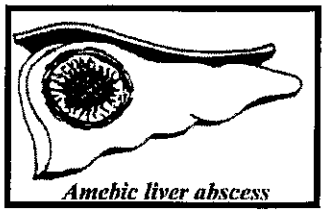
2. Recently percutaneous aspiration under abdominal U/S or CT guidance is done.

Amebic hepatitis and abscess

Incidence : Commoner than the pyogenic abscess in third world countries, particularly those lying in the tropical and the subtropical regions.

Etiology : Amebic hepatitis and abscess are due to infection with Entameba histolytica, as a complication of amebic colitis.

Mode of infection : Ingestion of cysts with improperly washed vegetables. In the intestine the cysts change into trophozoites that induce colitis.


Amebic liver abscess

Pathology :

- **The trophozoites** invade the portal blood from the colon.
- **The right lobe of the liver is affected more that the left,** as portal blood from the right side of the colon (site of amebic colitis), drains mainly to the right lobe of the liver while that from the left colon drains to the left lobe. The parasite starts a process of liquefactive necrosis which results in the formation of an abscess.
- **Number :** Usually single and unilocular.
- **Site :** Commonest in the posterosuperior segment of the right liver (as the segmental branch of the right portal vein to this segment is in direct continuity with the right portal vein).
- **The pus** is usually sterile with brown chocolate color, and is sometimes pinkish **(anchovy sauce).**
- **The wall** is shaggy and harbors ameba.

Complications :

I- Secondary bacterial infection : causing liver abscess.

II- Rupture :

[A] Upwards :

1. Subphrenic space : subphrenic abscess.
2. Pleural cavity : empyema thoracis.
3. Lung : amebic lung abscess.
4. Bronchus : expectorated contents with spontaneous cure or a bronchopleuro-biliary fistula develops.
5. Pericardium : pericarditis.

> • **When the abscess ruptures upwards, it usually leads to a lung abscess rather than empyema because the two layers of the pleura are usually obliterated.**

[B] Downwards : In stomach, duodenum, colon, renal pelvis or general peritoneal cavity leading to peritonitis.

[C] Outwards : rarely pointing below the costal margin.

[D] In the blood stream : pyemic amebic abscesses.

III- Destruction of the liver with liver cell failure and death.

IV- Calcification of a chronic liver abscess.

Clinical features :

[A] History : of amoebic colitis *(not always obtained)*

[B] General :

1. Low grade fever, anorexia, nausea, weakness, and weight loss.
2. The patient is usually pale and looks toxic **(earthy look).**

[C] Local :

1. Upper right abdominal pain.
2. There is usually tender hepatomegaly.
3. Tenderness in the lower right intercostal spaces.
4. The patient may have right sided pleural effusion and basal lung collapse.

[D] Picture of complications : (Discuss).

Differential diagnosis :

1. Pyogenic liver abscess.
2. Acute cholecystitis.
3. Perforated ulcer.
4. Acute viral hepatitis.
5. Pyelonephritis.
6. Hepatocellular carcinoma in black Africans assumes a rapidly progressive course with pain and constitutional symptoms that simulate those of an amebic abscess.

Investigations :

1. Blood picture : there is commonly leucocytosis and anemia.

2. Stool analysis.

3. Serological tests.

Positive 2, 3 are useful diagnostic aids in non endemic areas. In countries where amebic colitis is prevalent, these tests are likely to be positive in a high percentage of population.

4. Imaging by ultrasound and CT scanning : Can detect the number, site, and size of the abscess.

5. Chest X-ray : would show pleural effusion , pulmonary collapse & elevated right cupola of diaphragm.

6. Therapeutic test : Using Metronidazole.

An improvement in the general and local conditions after 3 days of treatment confirms the diagnosis

Treatment :

I- Conservative treatment : (Highly successful).
Metronidazole, 800 mg three times daily for 7-10 days is the drug of choice.

II- Ultrasound-guided percutaneous aspiration :

Indications :
1. Abscess that fails to respond to treatment within 72 hours.
2. Large abscesses.

Method :
- By a large probe needle under local anesthesia.
- **The site of aspiration :**
  > If anterior abscess : the needle is inserted below the costal margin.
  > If posterior abscess : the needle is inserted in the 10th intercostal space posteriorly.
- Few days later, ultrasound is repeated and if the abscess cavity has recollected aspiration is repeated.

If the abscess cavity has recollected and is more than 5 cm, aspiration is repeated

III- Open drainage :

Indications :
1. Presence of secondary infection.
2. If the abscess is pointing.
3. If aspiration is difficult because of multilocular abscess or the presence of thick pus.

Method :
Through the bed of the 12th rib, posteriorly, and through an extra-serous approach, as most of amebic abscesses are in the postero-superior segment of the right lobe.

Hydatid disease of the liver

Incidence : Prevalent is sheep-rearing parts of the world.
In Middle East, it is commonly seen in Iraq, Yemen, and Libya; but sometimes seen in Egypt.

Etiology :
- The disease is caused by tapeworm **Echinococcus granulosus.**
- The adult worm lives in the intestine of the dog **(definitive host).**
- Ova are passed in the dog's stools, and may be ingested by sheep **(2ry host),** once in the sheep's stomach, the ova hatch, penetrate the wall and enter the portal venous system to dislodge and form cysts mostly in the liver, and to a lesser extent in the lungs or brain.
- Dogs feed on liver of dead sheep.

Man may, by accident, become a 2ry host through vegetables or hands that become contaminated with dog's excreta.

Pathology of the cyst :
- **Number :** single or multiple.

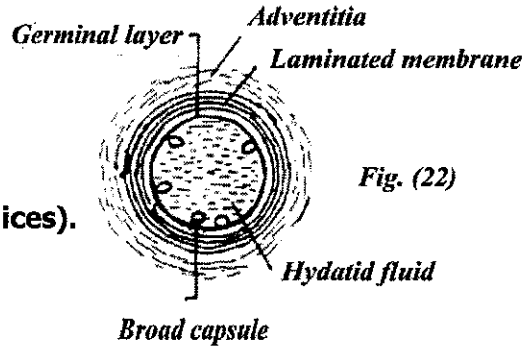


Fig. (22)

- **Structure :** Double-layer cyst wall.

1. The inner thin germinal layer **(endocyst)** shows many folds forming brood capsules that contain the heads of future worms **(Scolices).** This is the living part of the parasite that secretes the hydatid fluid.
2. The outer laminated membrane layer formed by the endocyst.
3. Adventitial fibrous tissue layer: formed by the host reaction.

- **The cyst fluid** is colorless and clear, but may be yellowish if there is a communication with bile ducts. Scolices are found in this fluid and may cause a severe anaphylactic reaction if it gets to the circulation.

- **Fate :**
  1. The majority of cysts continue to enlarge gradually.
  2. The parasite dies and the cyst is calcified.
  3. Complication :
     (a) Secondary infection.
     (b) Rupture in the biliary passages causing jaundice
     (c) Rupture in the peritoneal cavity causing dissemination and an anaphylactic reaction.

Clinical features :

1. Asymptomatic and discovered with Ultrasonography.
2. The usual presentation is by chronic right upper quadrant pain and hepatomegaly. In 70% of cases the cyst projects from the lower surface of the liver, forming a well defined **painless fluctuant swelling,** which often exhibits a **Hydatid thrill** on percussion due to the vibration of the daughter cysts.
3. Rarely, the disease presents by one of its complications.

Investigations :

1. Ultrasound or CT scan : shows the cyst (or cysts).
- In an endemic area, a liver cyst is usually caused by hydatid disease.

2. The intradermal Casoni's test :
- Disadvantages : 1. Once positive, it remains so for the rest of the patient's life.
  2. Gives false positive results in 40% of cases.

3. Hemagglutination, complement fixation tests and ELISA : detect hydatid antibodies.
- Advantages : 1. Most sensitive.
  2. Becomes -ve after death or treatment of the cyst.

4. Plain chest X-ray : To exclude associated chest affection.

Treatment :

I- Surgical removal :

- **Preoperative :**
  Estimation of the number and sites of the cysts is needed so as not to miss any of them.
- **The danger :** with hydatid cyst excision lies in the possibility of hydatid fluid spillage into the peritoneal cavity. This carries the double risk of :
  1. Forming other cyst.
  2. Absorption into the circulation inducing anaphylactic reaction.
- **Precautions :**
  1. The liver cyst (or cysts) is surrounded by dark green towels that are moistened with a scolicidal agent (hypertonic saline or betadine).
  2. The dark green color allows visualization of any spilled scolices.
- **Technique :**
  1. The cyst is first aspirated and hypertonic saline is injected.
  2. The overlying liver substance and the surrounding adventitia are incised, and the double layered cyst is easily enucleated.
  3. The cavity left by a large cyst is filled with pedicled omentum.

II- Medical treatment :

- Using **Mebendazole.**
- It is **not** a good substitute for surgery.
- **Indications :**
  1. Unfit patient. 2. Recurrent cases.
  3. Some authorities advise its routine postoperative use to prevent recurrence.
- **The dose :** 400 - 600 mg three times daily for one month.

Small asymptomatic cysts need just follow up

Some authors now advocate a policy of percautanous aspiration and injection of hypertonic saline or alcohol

Portal hypertension

Definition :

- The normal portal pressure equals 7 mmHg (8 - 12 cm of water).
- The average blood flow to the liver is about 1500 ml/min of which 2/3 come from the portal vein and 1/3 from the hepatic artery.
- Portal hypertension is present when the portal vein pressure exceeds 20 mmHg (25-30 cm of water).

Etiology :

I- Prehepatic causes :

1. Congenital malformation of the portal vein where it is replaced by cavernomatous mass.

2. Portal vein thrombosis secondary to :

(a) Neonatal umbilical sepsis. (b) Intra-abdominal sepsis. (c) Oral contraceptives.

II- Intrahepatic causes :

- Mainly due to liver cirrhosis and bilharzial periportal fibrosis.

  The causes of portal hypertension in cirrhotic patients are :

  1. Obliteration and compression of the sinusoids.
  2. Compression of the radicles of the portal and hepatic veins by the fibrous tissue.
  3. Arteriovenous shunts : between the branches of the hepatic artery and those of portal veins.

- **It is to be noted that in cirrhosis, the obstruction is sinusoidal and postsinusoidal while in bilharzial periportal fibrosis, the obstruction is presinusoidal.**

III- Posthepatic causes : (Rare)

1. Budd-Chiari syndrome which is caused by :

(a) Thrombosis of hepatic veins which may be spontaneous or secondary to polycythemia or the use of oral contraceptives.

(b) Malignant invasion of the hepatic veins.

2. Veno-occlusive diseases : in which there is obstruction of the hepatic venules.

Rule of 2/3 of portal hypertension :

- 2/3 of liver cirrhosis patients will develop portal hypertension.
- 2/3 of portal hypertension patients will develop esophageal varices.
- 2/3 of patients with esophageal varices will develop variceal bleeding.

Sequelae and clinical picture :

1. Porto-systemic collaterals :

Site	Between		Result
	Portal	Systemic	
1. The lower part of the esophagus and the fundus of the stomach	The tributaries of the left gastric and short gastric veins.	The esophageal veins which drain into the azygos and hemiazygos veins.	**Esophageal or gastric varices :** 1. Hematemesis. 2. Melena. 3. Fresh bleeding per rectum if the bleeding is massive.
2. The anterior abdominal wall	The paraumbilical vein if patent.	The veins of ant. abd. wall and superior & inferior epigastric veins.	**Caput medusae** and sometimes auscultation may detect a **venous hum** due to increased blood flow.
3. The lower rectum and anal canal	The tributaries of the superior hemorrhoidal vein.	The middle and inferior hemorrhoidal veins.	**Anorectal varices :** Due to congestion of the veins of the lower rectum and the anal canal.
4.Retroperitoneal	The tributaries of superior & inferior mesenteric veins.	The posterior abdominal wall and subdiaphragmatic veins.	Not important clinically.

2. Splenomegaly : Due to :

(a) Reticuloendothelial hyperplasia : due to absorption of bilharzial toxins.

(b) Venous congestion of the spleen which will be complicated by :

1. Recurrent attacks of splenic infarctions causing severe stitching pain and adhesions with the diaphragm and the anterior abdominal wall.
2. Secondary hypersplenism.

3. Congestion of the whole gastrointestinal tract leads to :

- Anorexia, Dyspepsia, Indigestion & Malabsorption.

4. The liver :

- **In Prehepatic causes :** usually normal.
- **In hepatic causes :** will show the pathology.
- **In Posthepatic causes :** congested liver.

5. Ascites : From :

(a) Hypoalbuminemia (nutritional or liver failure) is the main cause.
(b) Excess ADH and aldosterone (diminished hepatic destruction).
(c) Liver congestion and oozing of hepatic lymph (The Weeping Liver).
(d) Portal hypertension : acts to localize edema in abdomen (ascites) than in other parts of body.

N.B. It is extremely rare for portal hypertension alone to cause ascites.

Investigations :

- **The aims of investigations in a cirrhotic patient are :**

1. Assessment of the liver functions : Liver function tests will reveal :
(a) Hypoalbuminemia : The liver is the only site of albumin synthesis.
(b) ALT and AST are moderately raised.
(c) Prothrombin time and concentration are disturbed. This test is the most sensitive liver function.

2. Detection of esophageal varices : By either :
(a) Fiberoptic upper endoscopy.

Grading of esophageal varices:

Grade I: elevation of mucosa by dilated veins.
Grade II: elevated tortuous veins with normal mucosa in-between.
Grade III: no normal mucosa in-between.
Grade IV: impending rupture, e.g: erosion, ulceration, cherry-red spots.

(b) Barium swallow : they appear as multiple, smooth, rounded filling defects.
(c) Duplex scan can show dilated portal vein and collaterals.

N.B:

- ***The normal caliber of portal vein*** is up to 13mm,
- The ***normal caliber of splenic vein*** is up to 8mm (both increase in portal hypertension).

In Doppler study:

- the normal peak systolic velocity is 13-18 cm/sec. (decrease indicates portal hypertension).
- Normal direction of blood flow is ***hepatopetal*** (toward the liver) but it is reversed in potal hypertension (***hepatofugal***: away from the liver).

3. Detection of hypersplenism :

(a) Blood picture : will reveal anemia, leucopenia, thrombocytopenia (i.e. pancytopenia).
(b) Bone marrow examination : will reveal hypercellularity.
(c) Radioactive isotopes studies: using the patients own RBC's tagged with Cr51 will reveal diminished half life of RBC's and increased radioactivity over the spleen. The spleen / liver radioactivity index is increased (if more than 2:1 = hypersplenism).

4. Diagnosis of the pathology of liver disease : By :

(a) Immunological test : for hepatitis markers.
(b) Ultrasonography.
(c) Liver biopsy : after assessment of prothrombin time and concentration.

• To have an idea about the functions of the liver, **Child Pugh classification** can provide very useful data.

Child's Classification	1	2	3
1. Serum bilirubin (mg %)	Below 2	2 - 3	Above 3
2. Albumin	Above 3.5	3 - 3.5	Below 3
3. Ascites	None	Easily controlled	Poorly controlled
4. Nutritional status	Excellent	Good	Poor
5. Neurological disorders	None	Minimal	Coma

- **Patients with Child class A (5 - 7 points) classification can tolerate major surgery.**
- **Patients with Child class C (12 - 15 points) is not fit for major surgery.**

Modification of Child's classification :

1. Pugh modification (1973) : Dropped the clinical assessment of nutrition and included a further laboratory investigation the prothrombin time.

Prothrombin time (second prolonged)	A	B	C
	1 – 4	4 - 6	> 6

2. Hobb's modification : recommended prothrombin concentration instead of prothrombin time.

Other investigations which are less commonly used are :

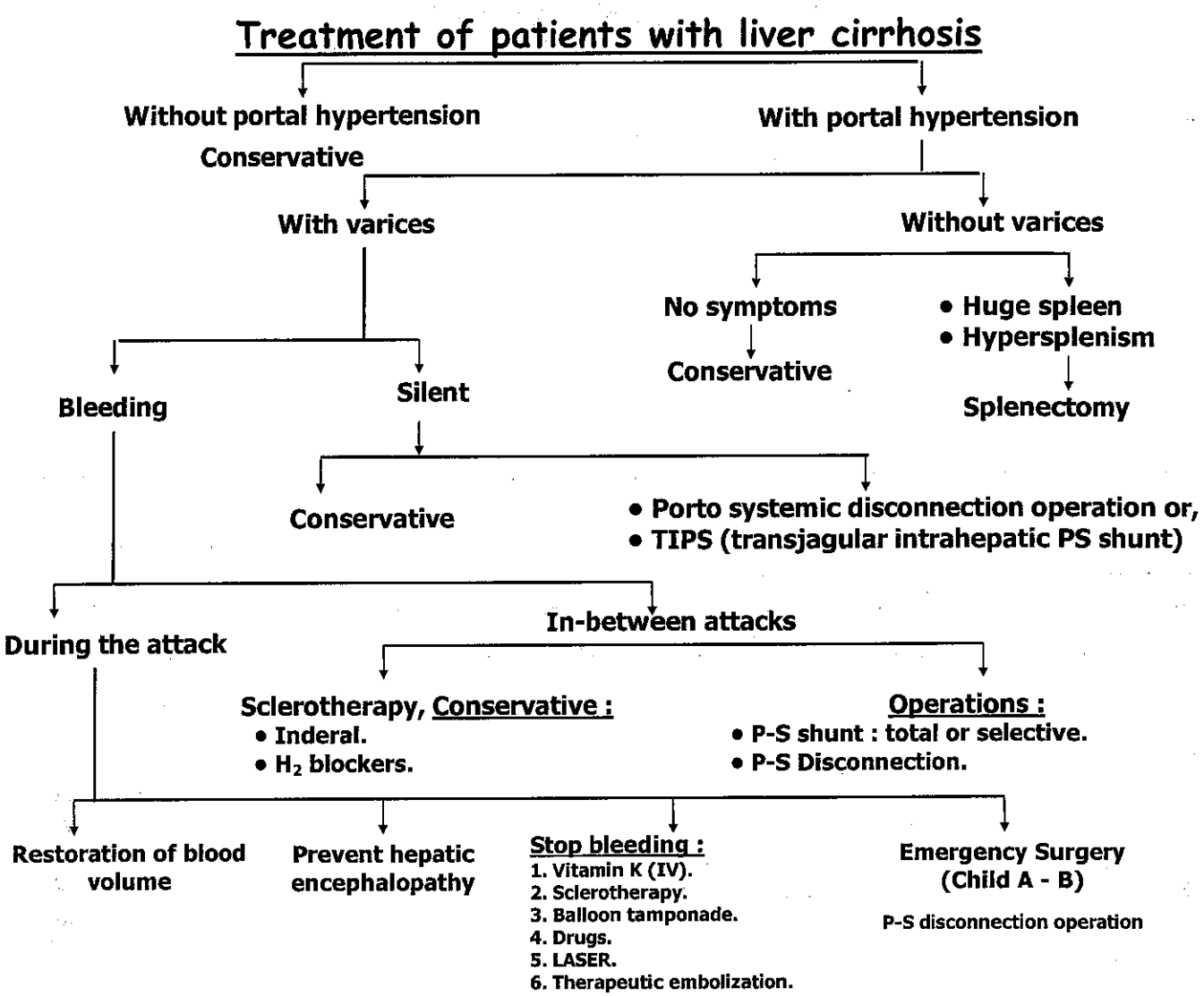
1. Portal manometry (measuring the portal pressure) :

(A) Pre-operative measurement :
1. Intrasplenic pressure by splenic puncture (presinusoidal pressure).
2. Occluded hepatic vein wedge pressure (sinusoidal pressure)

(B) During operation : Portal pressure is measured in any vein of the portal system.

2. Radiology :

(A) Splenoportography : to show whether the portal vein is obstructed or not.
(B) Venous phase of splenic artery or SMA angiography to show :
1. Patency of portal veins.
2. Anatomical variations.
3. Size of veins.
4. Grades of portal perfusion.



Treatment of patients with liver cirrhosis

I- Cirrhotic patients without portal hypertension : Conservative treatment.

II- Silent varices : Sclerotherapy is recommended if the varices are of large size or imminent to rupture on endoscopy.

III- Management of patients with active bleeding :

[A] Admission of the patient.

[B] Life saving resuscitative measures : should be initiated immediately and are then followed by diagnosis and definitive treatment.

[C] Correct coagulopathy : IV vitamin K.

[D] Prevention of hepatic encephalopathy : Every attempt should be made to evacuate blood from the gastrointestinal tract and to prevent its fermentation by :

1. Strong laxatives.
2. Gastric suction.
3. Repeated enemas or colonic lavage.
4. Oral lactulose : This is a disaccharide sugar which is fermented by the intestinal flora to lactic acid. The latter will combine with ammonia and it has a mild laxative effect.
5. Neomycin 0.5 gm every 4 hours can reduce the bacterial flora.

[E] Stop bleeding :

{1} Injection sclerotherapy :

- Should be performed as early as possible once the patient has been resuscitated.
- It is done under a mild sedative as diazepam.
- Done either intravariceal (5ml ethanolamine oleate into each varix) or perivariceal (0.5 ml aethoxysclerol are injected at each site). Both methods may be combined.
- In gastric varices intravariceal injection of histoacryl is performed.
- Injection must be repeated at 2 weekly sessions until the varices are obliterated.

- **Side effects of injection sclerotherapy :**
  1. Retrosternal discomfort for few days.
  2. Fever.
  3. Repeated injections can cause an esophageal ulcer which itself may cause bleeding.
  4. Repeated sclerotherapy may lead to fibrosis and stricture.
  5. Rarely penetration of the esophagus and mediastinitis.

{2} Drugs used :

1. Vasopressin :
- Action : vasoconstriction of the arterioles of the splanchnic circulation.
- Dose : 0.2 unit/kg wt. Dissolved in 200 ml of 5% dextrose and given over 20 minutes.
- Disadvantages : colicky abdominal pains, diarrhea and anginal pains.
- Contraindicated : in the elderly.

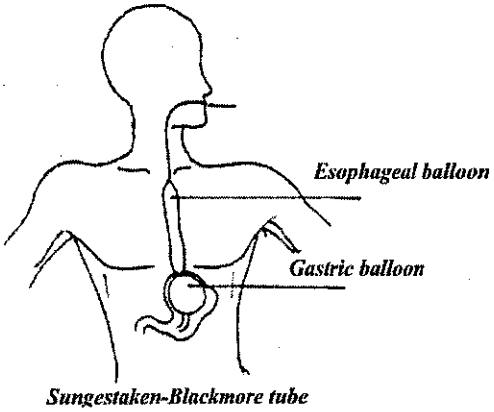
2. Somatostatin : can lower the intra variceal pressure but is very expensive.

3. Glypressin :
- Synthetic derivative which has the same hemodynamic effects as vasopressin but it controls variceal bleeding better than vasopressin.
- Dose : 2 mg / 6hrs IV maximum for 24 hours.

{3} Balloon tamponade :

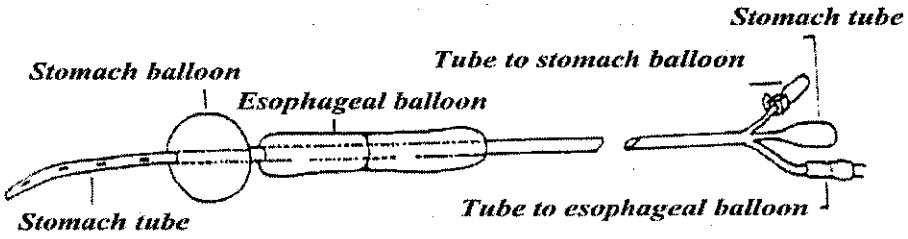
- **By Sungestaken-Blackmore or Linton tube.**
- **Method :** The gastric balloon is inflated by 200 ml of air then pulled upwards to press the fundus.
- **If the bleeding continues,** the esophageal balloon as well is inflated. The pressure in the esophageal balloon should not exceed 40 mmHg.

- **Disadvantages :**
  1. Discomfort to the patient.
  2. The patient cannot swallow his saliva.
  3. Liability to cause esophageal ulceration or stricture.
  4. Re-bleeding in 60 - 90% of patients.
  5. Ulceration of the nostril.



Sungestaken-Blackmore tube

Balloon tamponade is only used as a temporary measure before sclerotherapy or surgery



- **Tubes other than Sungestaken :**

1. Minnesota 4 lumen tube : the 4th tube to aspirate saliva from the esophagus around the esophageal balloon.
2. Linton tube : has only the gastric balloon.

{4} Band ligation :

- The idea is to encircle each varix by a tight band which leads to thrombosis of the veins.
- It is as effective as sclerotherapy in control of bleeding but with less number of sessions.
- It causes some discomfort and it may produce some lacerations.

{5} Infra-red photocoagulation :

- **Indications :** The same as injection Sclerotherapy
- **The idea :** Infrared photocoagulation can give a tissue temperature of 100°C which produces an area of coagulation necrosis. The dead tissues separate after 10-14 days leaving a granulation lined ulcer.
- This is an effective and painless method.

IV- Emergency surgery : Porto-systemic Disconnection operations :

Indications :

1. If all the previous measures fail to stop bleeding.
2. Satisfactory general condition of the patient.

Methods :

1. Splenectomy, Portoazygos Disconnection and Stapling of the esophagus : (Child A-B).
2. If the patient is not very fit : Stapling alone can be performed.
N.B: bleeding from varices is related to: size of varices, bad liver functions (child C), hepatic vein wedge pressure more than ***10 mmHg***.

V- Treatment of patients with history of bleeding esophageal varices :

- Repeated sclerotherapy until the varices are obliterated.
- Inderal proves to lower the portal pressure & is helpful in such patients (The general trend nowadays).

N.B:recently ***percutaneous transhepatic obliteration of varices*** is tried in poor risk patients: under US guidance needle is inserted percutaneous transhepatic and introduced to the portal vein then to left gastric vein with injection of sclerosant material (thrombin or gel foam).

Surgery : Indications :

1. If sclerotherapy failed to stop recurrent attacks of bleeding.
2. Fit patient (Child A-B).

Operations for portal hypertension

(1) Shunt operations

[A] Total shunt operations

Shunt the whole portal blood to the systemic circulation.

1. Porto-caval operation :

- The distal end of the portal vein is divided and transfixed while the proximal end is anastomosed to the anterior surface of the IVC.

Advantages : very efficient in lowering the portal pressure.

Disadvantages :

1. Deprives the liver of the portal blood flow and this accelerates the onset of liver failure.
2. Shunting the portal blood completely away from the liver leads to hepatic encephalopathy in up to 50% of patients.

2. Proximal spleno-renal shunt :

Splenectomy is performed and the proximal end of the splenic vein (the side towards the portal vein) is anastomosed to the left renal vein.

Indications :

1. If the portal vein is thrombosed.
2. If splenectomy is indicated.

Advantages : the incidence of encephalopathy is less than after porto-caval shunt.

Disadvantages :

1. Less effective in preventing further bleeding.
2. The anastomosis is more liable to thrombose.

3. Mesocaval shunt :

- Comprises insertion of a graft (synthetic or vein) between the superior mesenteric vein and the IVC.

Disadvantages : The incidence of thrombosis is high.

[B] Selective shunt operations

1. Warren shunt: distal lieno-renal shunt:

Shunt the portal flow away from the area of lower esophagus and upper stomach :

- The right and left gastric vessels are ligated.
- The proximal end of splenic vein is ligated while the distal end is anastomosed to the left renal vein.
- The short gastric veins are preserved and will selectively decompress the lower end of the esophagus.

Advantages : The incidence of encephalopathy is low, and the liver functions remain normal.

Disadvantages: pancreatic siphon action: by time it turns into total shunt due to presence of connections into the pancreas between splenic and portal vein.

2. Modified Warren shunt: distal part of splenic vein is freed from pancreas to avoid pancreatic siphon.

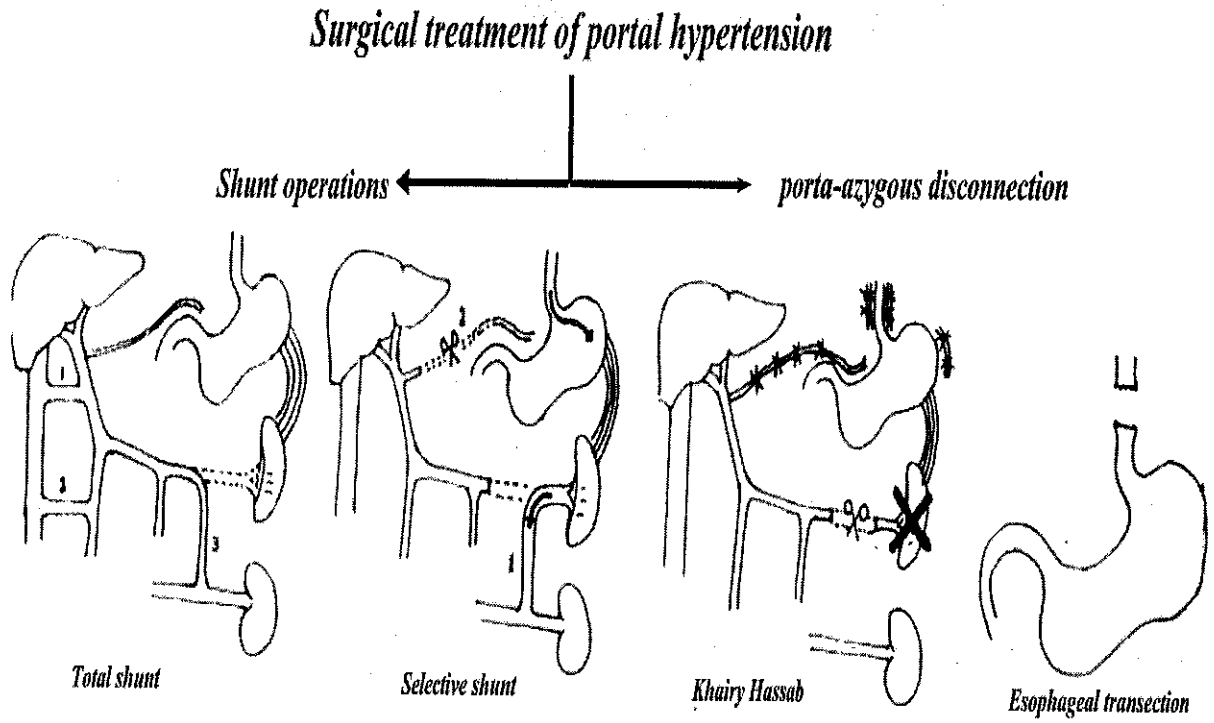
3. Inokuchi shunt: shunt between left gastric vein and inferior vena cava using a graft (autogenous or synthetic)

N.B.: TIPS : (Transjugular Intrahepatic Portosystemic Shunts)

A special stent is passed through internal jugular vein to the hepatic vein to create a Portosystemic shunt.

- **Indications :**

1. In case of severe liver cirrhosis before liver transplantation.
2. Refractory ascitis.



(2) Porta-azygos disconnection operations

[A] Hassab Khairy operation : Entails :

1. Splenectomy.
2. Ligation of the right and left gastric vessels and the short gastric vessels leaving only the right gastro-epiploic vessels.
3. All vessels surrounding the lower 5-10 cm of the esophagus are ligated.

Advantages :

1. No encephalopathy.
2. Portal blood flow is intact.
3. Low incidence of re-bleeding following the operation, which can usually be controlled by sclerotherapy.

[B] Esophageal transaction : Entails :

- Interruption of the submucosal venous plexus which is still intact following porta azygos disconnection.

Advantages : simple and easy and it can be added to porta azygos disconnection.

[C] Sugiura shunt: Done transabdominally with:

- Esophageal transection
- Splenectomy
- Devascularization of the proximal part of the stomach
- vagotomy and pyloroplasty.

(3) Liver transplantation

- With end stage liver disease.

Treatment of ascites

[A] Medical treatment :

- Initial treatment with spironolactone (200 mg/day).
- If spironolactone alone is insufficient another drug such as frusemide should be added.

[B] Tapping :

- Ascites of liver failure (bad liver function) resists all forms of treatment.
- Paracentesis (tapping) may be done if there is respiratory distress.

[C] Surgical treatment :

1. Total porta-caval shunt : provided that the portal pressure is high & the liver functions are not bad.
2. Le Veen shunt : It is indicated in refractory ascites. A subcutaneous silastic catheter is connected to the peritoneal cavity and the jugular vein. It transports ascitic fluid from the peritoneal cavity to the jugular vein. It has a small unidirectional valve sensitive to a pressure gradient of 3-5 cm water which prevents back flow of blood.
3. Lympho-venous shunt.

Tumors of the liver

Benign	Malignant	
Liver cell adenoma	**Primary**	**Secondary**
• The use of CCP is involved in its initiation & progress. • Cessation of pill intake can result in tumor regression.	1. Hepatocytes : Hepatocellular Carcinoma (HCC).and hepatoblastoma 2. Bile Ducts: Cholangiocarcinoma. 3. Mixed: Hepatocholangiocarcinoma.	From : 1. GIT. 2. Pancreas. 3. Breast.

Non-neoplastic lesions of the liver (similar to benign tumors)
1. ***Hemangioma.*** : It is the commonest benign tumor of the liver and the liver is the commonest visceral organ affected by hemangioma. **Treatment:** is usually just follow up, rarely localized resection or hepatectomy is indicated.
2. ***Focal nodular hyperplasia.***

Primary Malignant Liver Tumors:

1. Hepatocellular carcinoma

Incidence: Commoner in males.
Prevalent in the far east and equatorial African nations.
The highest world's incidence of primary liver cancer is in Mozambique.

Etiology :

1. Infection with hepatitis B virus **(HBV)** or hepatitis C virus **(HCV).**
   a) **Hepatitis B:** there is integration between DNA of the virus and DNA of liver cells leading to abnormal proliferation of hepatocytes.
   b) **Hepatitis C (RNA virus):**hepatoma occurs secondary to cirrhosis by chronic irritation.
2. Liver cirrhosis, highest incidence is with post-hepatitic, followed by alcoholic cirrhosis.
3. Aflatoxin ingestion. The fungus Aspergillus flavus that grows on grains that are stored in moist warm conditions forms this substance. The poor storage conditions of grains in Africa favor its growth.
4. Hepatic adenoma:rare.

Pathology :

Gross appearance: The tumor is yellow in color and commonly arises in a cirrhotic liver.
- It assumes one of different appearances:

1. Massive form i.e. forming a localized mass. 2. Nodular form. 3. Diffuse form.

Microscopically : HCC is formed of malignant hepatocytes and stroma, with high vascularity.

Complications :

1. Spread by the lymphatic and venous routes producing porta hepatis nodal enlargement, peritoneal nodules & less commonly lung deposits.
2. Spontaneous rupture that may produce massive intra-peritoneal hemorrhage.

2. Cholangiocarcinoma

- It has its highest incidence in the Far East.
- The tumor is an adenocarcinoma. It arises from the intra-hepatic bile ducts.
- Its development is related to infestation with the liver fluke clonorchis sinensis that causes ascetic cholangitis.

3. Hepatocholangiocarcinoma

- This is a mixed type that behaves like a HCC.

Clinical picture of Primary Malignant Liver Tumors:

- **There are different types of presentation :**

1. A common presentation is the deterioration of health of a known cirrhotic.

2. Small lesions may be discovered through an abdominal ultrasound examination that is done for another purpose.

3. In areas where HCC is a common tumor an early detection program is recommended where known cirrhotics have an annual ultrasound examination and α-fetoprotein estimation. An elevation of the tumor marker level or the appearance of a focal lesion warrants further investigations to detect the tumor at its earliest phase.

4. HCC in black Africans grows rapidly and degenerates producing pain, jaundice & fever. It also produces a tender mass, which is commonly mistaken for amebic liver abscesses.

5. Late cases present by pain, jaundice, ascites, Hepatomegaly, anorexia, loss of weight and probably massive intraperitoneal hemorrhage.

6. Paramalignant syndromes e.g.hypercalcemia,fever,hypoglycemia,polycyathemia.

NB. Fibrolamellar HCC is a variety of the massive type of HCC that affects non-cirrhotic young females, and hence has a better prognosis.

Investigations :

[A] Laboratory tests :

1. Serum alkaline phosphatase is often elevated. The rest of liver function tests may show the pattern of cirrhosis if present.
2. Serum α-fetoprotein is a tumor marker.

Alpha-feto protein

- Highly elevated with HCC & with testicular teratoma.
- The normal adult level is zero to 10ng/ml.
- Level above 200ng/ml is suggestive for HCC.
- Level above 2000ng/ml is diagnostic for HCC.
- Returns to normal level after a successful resection.
- Re-elevation in the follow up period signifies a recurrence.

[B] Imaging :

1. Ultrasound, CT scan or MRI.
2. Hepatic angiography.
3. Chest x-ray and CT scan to search for chest metastases.
4. Laparoscopy may be done to visualize the extent of the tumor and to perform laparoscopic U/S.
5. **The value of preoperative guided biopsy is controversial because of the possible risk of hemorrhage from the highly vascular tumor.** ***Preoperative guided biopsy is not desirable.***

- Intra-operative ultrasound allows safer liver resection. it is used to detect small lesions, which may be missed by other imaging.
- Triphasic multi-silice CT scan via I.V contrast is mandatory to show relations of tumour to liver vasculature.
- Laparoscopy may be done to visualize the extent of the tumor and to perform laparoscopic U/S.

Treatment :

Liver resection is the only hope for cure, but unfortunately, it is suitable for a small proportion of cases.

[A] Operable cases (tumor localized to one liver lobe) :

- The usual operation for such situation is either right or left hepatectomy.
- The presence of cirrhosis in most cases poses some problems due to :
  1. Bleeding tendency.
  2. Poor function of the remaining half-life.
  3. Diminished capacity of liver cell regeneration.

[B] Inoperable cases :

1. Systemic chemotherapy.
2. Intra-arterial selective chemotherapy via a catheter inserted in the hepatic artery.
3. Chemoembolization : the artery is blocked producing tumor ischemia & the drug is selectively delivered to its target.
4. Percutaneous ethanol injection.
5. Percutaneous P53 (tumor suppressor gene) injection
6. Percutaneous thermal ablation by radio-frequency
7. Percutaneous laser therapy.
8. Percutaneous cryo-surgery.

Liver metastases

Incidence : 20 times more common than primary malignancy.

Metastases can reach the liver through either :

1. Portal circulation : This is the commonest route. Carcinomas of the colon, rectum, pancreas, stomach or small bowel metastasize primarily to the liver.

2. Hepatic artery : Carcinomas of the lung, breast, kidney, uterus or ovaries are common examples.

3. Lymphatics.

4. Direct spread : from tumors of the gall bladder, stomach, or colon.

Pathology :

- May occur synchronously or metasynchronously to the primary tumor.
- Metastatic nodules are usually multiple, white and umbilicated because of central necrosis.
- Over 90% of patients with hepatic metastases have tumor deposits in other organs.

Clinical features :

1. Picture of the primary.
2. Weight loss, fatigue and anorexia.
3. Right upper abdominal pain.
4. Jaundice.
5. Ascites.
6. Palpable hepatomegaly.

Differential diagnosis of focal hepatic lesion

1. HCC
2. Metastasis
3. Benign lesion Hemangioma, adenoma and focal nodular hyperplasia
4. Cyst (parasitic or non parasitic)

Investigations :

1. Serum alkaline phosphatase and bilirubin are usually elevated.
2. Imaging by ultrasound, CT scan or MRI.

Treatment :

Generally, the presence of liver metastases indicates an advanced inoperable tumor. The treatment in such cases (usually by chemotherapy) is considered palliative.

- **Few selected cases of metastatic colorectal cancer, if localized to one segment, can be treated by liver resection aiming at cure.**

[A] Chemotherapy :

Systemic, selective intra-arterial chemotherapy through a catheter that is surgically inserted in the hepatic artery.

[B] Liver resection : Requirements :

1. A completely resectable colorectal cancer with no apparent residual tumor.
2. No extrahepatic metastases.
3. A solitary metastatic nodule or multiple metastases that are confined to one surgical lobe of the liver.

- **Liver resection is done in the same session of the colorectal operation, or later if the liver deposits appear in the course of the patient's follow up.**

Liver call adenoma (LCA)

- It is a true benign neoplasm of hepatocytes
- The use of contraceptive pills is incriminated in its initiation and progress. Cessation of pill intake can result in tumour regression.

Incidence : It occurs almost only in women.

Pathology:

Grossly: LCA is soft, well circumscribed. It is multiple in 1/3 of cases.

Histologically: LCA is formed of sheets of regular hepatocytes with thin-walled vessels.

Clinically: the tumour is commonly asymptomatic and is discovered incidentally at U/S examination or at laparotomy. Sometimes a large LCA causes right upper quadrant pain.

Investigations:

- Imaging by ultrasound, CT scan and/or MRI.
- Liver function tests and alpha-feto protein are within normal levels.

Complications: rare and include malignant transformation and spontaneous rupture.

Treatment:

- Liver resection is indicated if doubt exists as to the possibility of malignancy and for large symptomatic tumours.
- In other cases, OCPs intake is discontinued and the tumor is regularly checked for change of size.

Focal Nodular Hyperplasia (FNH)

- This is a harmless lesion of the liver.
- Its main importance is the difficult differentiation from other focal lesions.
- Contraceptive pills stimulate the development and the growth of FNH.

Macroscopically: FNH is a nodular firm mass, which is slightly paler than the normal liver and has prominent surface vasculature. The cut section shows a satellite scar with radiating fibrous septa that divide the lesion into lobules.

Histologically: it resembles cirrhosis with regenerating nodules.

Diagnosis:

- The pathognomonic satellite scar can be seen on CT scan.
- Sulphur colloid scan; it appears as hot area, this differentiates it from HCC or metastases.

Treatment: Most cases require no treatment. Contraceptive pills should be discontinued.

Haemangioma

- Hemangiomas are the commonest benign tumours of the liver.They may attain a large size.
- They are usually harmless, but they occasionally rupture producing serious bleeding.
- Contrast suspicion of a Hemangioma is a contraindication for biopsy.
- Asymptomatic Hemangiomas that are diagnosed at laparotomy should not be resected as the natural history is towards involution and calcification.
- Surgical excision is reserved for the rare lesions that produce pain or bleeding.

Regarding Liver biopsy

- The standard method is the per-cutaneous technique. The commonly used needles are the aspiration (Menghini) and the cutting (true cut) types. PT should not be prolonged more than 3 times.
- Hemangiomas & hydatid cysts should not be biopsied

Causes of Splenomegaly & Hepatomegaly

	Causes of Splenomegaly	Causes of Hepatomegaly
Congenital	1. Cysts of the spleen.	1. Reidel's lobe. 2. Polycystic disease.
Infection	**1. Bacterial :** Typhoid, Paratyphoid, Brucellosis, Tuberculosis, Typhus, Anthrax, Abscess & Syphilis.	**1. Bacterial :** Syphilitic gumma, Leptospirosis, Typhoid, Brucellosis, Pyogenic Cholangitis, Pyelophlebitis & Portal pyemia.
	2. Viral : Infectious Mononucleosis & Psittacosis.	**2. Viral :** Infectious Mononucleosis, Viral hepatitis, CMV, Herpes simplex.
	3. Parasitic : Bilharziasis & Hydatid cyst.	**3. Parasitic :** Bilharziasis, Hydatid cyst, Toxoplasmosis & Kala-azar.
	4. Protozoal : Kala-azar and malaria.	**4. Amebic hepatitis.** **5. Toxoplasmosis.**
Blood Diseases	1. Leukemias. 2. Hemolytic anemias. 3. Polycythemia vera. 4. Myelofibrosis.	1. Leukemia (lymphatic and myeloid). 2. Hemolytic anemias. 3. Polycythemia vera. 4. Myelofibrosis.
	5. Thrombocytopenias. 6. Thalassemia major.	5. Megaloblastic anemia.
Metabolic Causes	1. Amyloidosis. 2. Gaucher's disease. 3. Porphyria and rickets.	1. Amyloidosis. 2. Hemochromatosis. 3. Wilson's diseases. 4. Fatty liver e.g. DM, obesity, Reye's syndrome. 5. Lipid storage diseases. 6. Glycogen storage disease e.g. Von Geirke's disease.
Circulatory	**Portal vein occlusion :** 1. Portal hypertension. 2. Neoplastic lesions e.g. cancer body and tail of pancreas. 3. Thrombophlebitis.	**1. Chronic venous congestion :** CHF, pericardial effusion and Constrictive pericarditis. **2. Portal hypertension :** from hepatic and post-hepatic causes e.g. Budd-Chiari syndrome & veno-occlusive disease.
Tumors	**Benign :** 1. Hemangioma. 2. Lymphangioma.	**Benign :** 1. Hemangioma.
	Malignant : 1. Fibrosarcoma. 2. Malignant lymphoma. 3. Waldenstrom macroglobinemia.	**Malignant :** 1. Metastases. 2. Malignant lymphoma. 3. Hepatoma. 4. Cholangioma.
Collagen Diseases	1. Felty's syndrome Stills disease. 2. Sarcoidosis.	1. Felty's syndrome Stills disease. 2. Sarcoidosis.

Causes of enlarged tender liver (Rapid liver enlargement)

1. Chronic venous congestion (CHP and constrictive pericarditis).
2. Amebic hepatitis and amoebic liver abscess.
3. Viral hepatitis.
4. Pyogenic liver abscess.
5. Malignancy.

Spleen

Rupture of the spleen

Incidence :

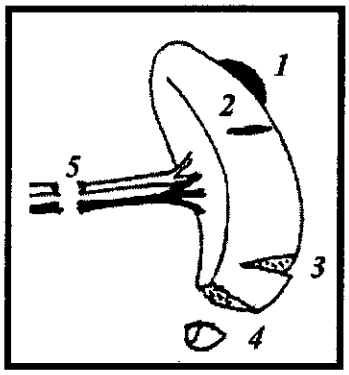
The commonest solid abdominal organ to be injured in blunt abdominal trauma.

Predisposing factors :

1. **Splenic enlargement** which makes it more liable to trauma.
2. **Diseases of the spleen** like malaria which make it soft and more friable.

Types of trauma :

1. **Penetrating trauma :** e.g. gunshot wounds or punctures due to stabbing or missile.
2. **Blunt abdominal trauma or trauma to the lower thoracic cage :** e.g. road traffic accident or falling from a height.
3. **Spontaneous rupture of the spleen** is rare.
4. **Operative trauma :** e.g. during gastric or colonic surgery.



Pathology : The types of rupture may be :

1. Subcapsular hematoma.
2. Small superficial tears, single or multiple.
3. Deep tear, single or multiple reaching the hilum.
4. Avulsion of a pole of the spleen.
5. Complete pulping of the spleen.
6. Injury of the vascular pedicle, i.e. avulsion or thrombosis of the vessel.

Complications :

1. Hemorrhage which may be :

(A) External hemorrhage if it is an open injury.

(B) Internal hemorrhage which may be intraperitoneal, extraperitoneal (retroperitoneal hematoma) or intrapleural if the injury involves the diaphragm.

Associated other abdominal or thoracic injuries.

Splenic cyst : may follow a perisplenic hematoma.

Clinical picture : There are three clinical types of rupture of the spleen.

Fatal type

The tear is deep or the pedicle is ruptured and hemorrhage is massive, so the patient is severely shocked with rapid death before any surgical intervention.

Classical type

This is the commonest presentation in which there are :

1. Shock.
2. Recovery from shock.
3. Manifestations of internal hemorrhage with increase in pallor, weak and rapid pulse, low blood pressure and air hunger.

Physical examination :

- **Tenderness and rigidity** in the left hypochondrium which spreads into the abdomen.
- **Evidence of shifting dullness.**

Special signs : may be present in rupture of the spleen but they are not essential for the diagnosis :

1. Balance's sign : Shifting dullness on the right side and fixed dullness on the left side, this is due to free fluid blood in peritoneal cavity on right flank but on the left flank the fixed dullness is due to retroperitoneal hematoma.

2. Kehr's sign : The patient has pain in the left shoulder which is referred from irritated diaphragm. This pain occurs especially if the patient is put in the Trendlenburg position. Also pressure on the left hypochondrium leads to referred pain in left shoulder.

3. Cullen's sign : Brownish or bluish discoloration around the umbilicus may occur.

Delayed rupture

The initial shock is followed by long lucid interval which may be few days or weeks after the accident, which may be forgotten, the patient presents with clinical picture of internal hemorrhage.

This delay may be due to :

1. The formation of subcapsular hematoma which may rupture later.
2. The greater omentum seals the region of the spleen from the general peritoneal cavity, and then retracts releasing blood.
3. A clot may form to block the tear and stop bleeding and later on it is displaced when the blood pressure rises or it is digested by enzymes from the injured pancreas.

Investigations :

1. Blood picture.

2. Ultrasound : Will show :

- The spleen with a surrounding hematoma.
- Free blood in the peritoneal cavity.

3. Plain X-ray of the upper abdomen : May show :

- Obliteration of psoas shadow.
- Indentation of left side of gastric air bubble.
- Fracture of one or more of lower ribs (in the left side).
- Elevation of left side of diaphragm.
- Obliteration of splenic outline.

4. CT scan : of the abdomen.

5. DPL : Reveals blood.

If the patient is in severe shock there is no need for investigations.

Treatment :

1. Remember ABCDE.

2. Correct the hypovolemic shock by crystalloid and blood transfusions.

3. Laparotomy : Rapid exploration through a midline incision is performed :

- In the majority of cases splenectomy will be the procedure of choice.
- Any associated injury is dealt with.

4. In children especially below the age of 15 years every attempt should be made to preserve the spleen due to the vital role it plays in the immune mechanism in this age group.

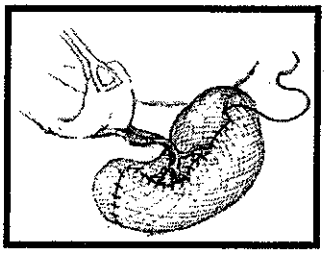
Several techniques for splenic preservation are available :

1. Splenorrhaphy : using vertical mattress sutures & hemostatic adhesive.

2. Partial splenectomy : excision of the damaged segment following ligation of the appropriate segmental artery.

3. Splenic wrapping procedures :

- A window is made in a mesh to accommodate the splenic artery & vein.
- The spleen is enveloped by the mesh by suturing the free edges of the mesh.



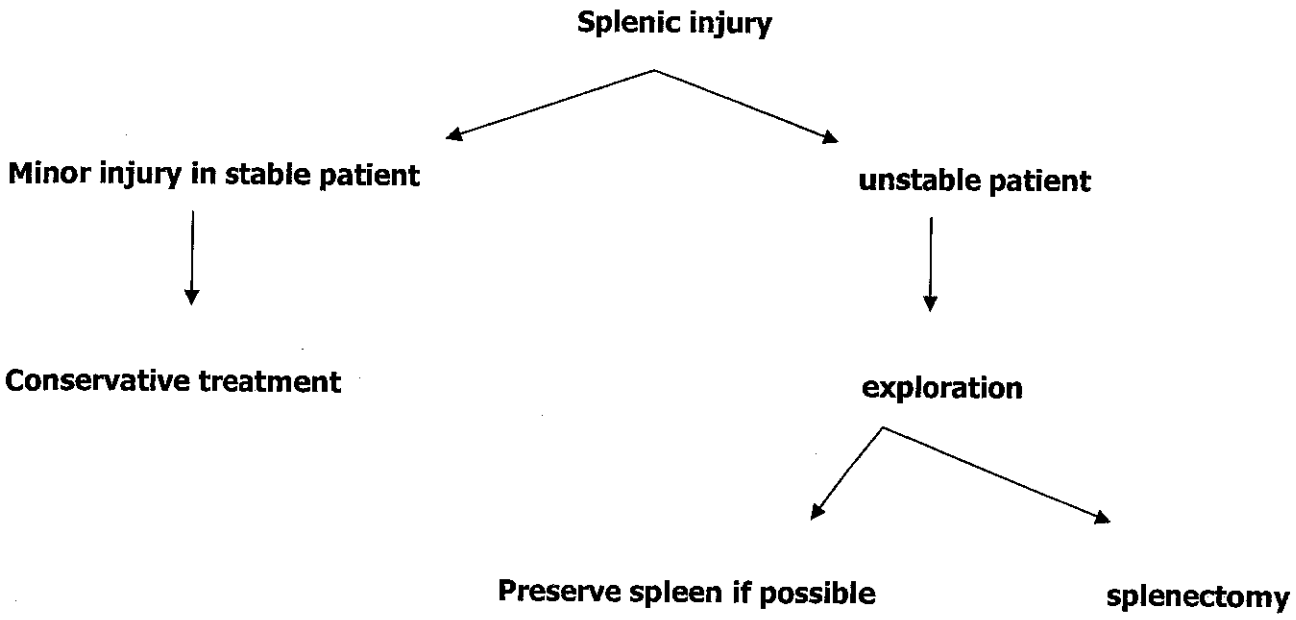
4. Autotransplantation of fragments of splenic tissue wrapped by omentum.

5. Ligation of splenic artery.

6. Therapeutic embolization by gel foam of splenic artery.

- **If conservative surgery is chosen meticulous post operative observation is essential.**
- **If splenectomy is necessary in a young child it is recommended to administer pneumococcal vaccine (pneumovax) cover until 18 years of age.**

Algorithm summaries the treatment of splenic injuries



Infections of the spleen

Acute abscess :

- This is a rare condition occurring as a sequel of specific fever, e.g. typhoid fever.
- It can also be blood borne from a distant systemic pyemia.
- The treatment is splenectomy if possible, but if there are dense adhesions, drainage of the abscess is only performed.

2. Chronic abscess : Treatment is by splenectomy

3. Tuberculosis : The disease is usually a manifestation of generalized miliary TB.

4. Syphilis of the spleen : is now rare.

5. Parasitic infection of the spleen : occurs in schistosomiasis and hydatid cysts.

6. Protozoal infection of the spleen : occurs in malaria and Kala-azar (due to Leishmania donovani).

Bilharziasis of the spleen (Egyptian Splenomegaly)

Etiology :

- Schistosoma mansoni in 75% of cases.
- Schistosoma hematobium in 25% of cases.

Incidence :

Affects farmers (any age) of Lower Egypt and males are more commonly affected.

Causes of splenomegaly in bilharziasis :

1. Reticulo-endothelial hyperplasia due to : (early)

(a) Absorption of toxins from the living worms and ova.

(b) Deposition of ova in the splenic radicles.

2. Congestive splenomegaly. (late)

Pathology :

Gross picture :

- The spleen is enlarged with a thick capsule.
- Subcapsular hemorrhages & patches of perisplenitis, due to organized hematomas or infarcts.
- Thick vascular adhesions may bind the capsule to the diaphragm and nearby structures.
- The splenic vessels are markedly enlarged and tortuous.
- On section the cute surface is firm in consistency and red in color with prominent lymphoid follicles and thick greyish trabeculae.

Histologically :

- The red pulp is congested and infiltrated with eosinophils.
- The lymphoid follicles are enlarged and may show germinal centers.
- The littoral cells of sinusoids & reticular cells of the pulp proliferate & show active phagocytosis.

Clinical picture	Complications	Stages
• Picture of liver cell insufficiency and portal hypertension. • **Syndrome of :** 1. Pain : > Dragging pain if very large spleen. > Stitching pain if infarction occurs. 2. Bilharzial colitis. 3. Liver cirrhosis. 4. Splenomegaly. 5. Irregular fever. 6. Pancytopenia : Anemia, leucopenia and thrombocytopenia in hypersplenism.	1. Susceptibility to injury. 2. Susceptibility to infarction. 3. Hypersplenism.	1. Hepatomegaly. 2. Hepatosplenomegaly. 3. Splenomegaly with shrunken liver. 4. Ascites. 5. Liver cell failure.

Treatment :

Splenectomy in patients with Egyptian hepatosplenomegaly only in the following conditions :

1. Hypersplenism.
2. Huge splenomegaly.
3. Recurrent attacks of pain secondary to splenic infarctions.
4. As a part of operations performed for portal hypertension as porta-azygos disconnection or splenorenal shunt.

• It may be difficult or even impossible to do splenectomy as adhesions may bind the capsule to the diaphragm and nearby structures.

Congenital anomalies of the spleen

1. Agenesis : absence of the spleen is very rare.

2. Accessory spleens :

- Occurs in 10-35% of individuals.
- The majority of accessory spleens occur at the hilum, in the greater omentum or along the splenic vessels and pancreas.
- If the spleen is removed for hemolytic anemia, a careful search for accessory spleens which may cause recurrence of symptoms if not removed.

3. Polysplenia : the normal spleen is deeply lobulated.

4. Splenic cysts : dermoid & mesenchymal inclusion cysts.

5. Wandering spleen (splenic ectopia) : the spleen is freely moving in the abdomen due to laxity of gastrosplenic and splenophrenic ligament. Torsion of the pedicle is the most frequent complication.

Cysts of the spleen

Multiple :

- Cystic disease of the spleen is usually associated with cystic disease of liver, kidney, lung and pancreas.

Single :

- Hydatid cyst, lymphatic cyst, blood cyst or cystic degeneration of tumors.

Splenic Tumours

- They are not common.
- They may be
  1. Part of part of reticulo-endothelial hyperplasia as in malignant lymphoma
  2. tumours confined to spleen:
     a. **Benign**: Lymphangioma & cavernous Hemangioma.
     b. **Malignant**: localized form of lymphoma and fibrosarcoma.
     c. Spleen may be infiltrated by direct extension from carcinoma of the stomach or pancreas. **Malignant Metastasis is very rare**, it is supposed that RE cells destroy them.

Infarction of the spleen

- **Causes**
  - It may be spontaneous in splenomegaly
  - Vascular occlusion by sickle cell disease
  - Embolism from infected heart valve
- It may be asymptomatic but commonly presents with left upper quadrant abdominal pain & guarding
- The patient needs rest & analgesics. Splenic abscess requires splenectomy.

Functions of the spleen

1. Fetal erythropoeisis: during the 5^{th} -8^{th} month during fetal life

2. Sequestration of old blood cells

3. Defensive functions:

a) Reticulo-endothelial macrophages of the spleen destroy foreign substances.
b) Proliferation of T and B lymphocytes.
c) Production of antibodies e.g. IgM,Opsonins,tuftsin,properdin and interferons.

Indications for splenectomy

1. Traumatic injury of the spleen.

2. Hematological diseases :

(a) Hereditary spherocytosis.

(b) Idiopathic thrombocytopenic purpura. Indicated if manifestations persist for more than 9 months

(c) Some patients with acquired hemolytic anemia.

(d) Some patients with Thalassemia.

3. Lymphomas : Splenectomy is performed for staging & to reduce the dose of radio or chemotherapy.

4. Portal hypertension.

5. Hypersplenism.

6. Splenic cysts, abscess or tumors.

7. During radical surgery for the stomach, esophagus or pancreas.

Indication of Splenectomy In Hemolytic Anemia

1. Heriditary spherocytosis is cured by splenectomy. Best time between 6-7 years old.
2. Some cases of thalassaemia require splenectomy, which is less beneficial than in spherocytosis.
3. Rarely sickle cell anemia or autoimmune types.

N.B: Regarding hereditary spherocytosis

a. Pigmented gall stones may occur in up to 60% of patients; may occur under 10 years old.
b. If the patient has gallstones, cholecystectomy can be performed simultaneously.

Complications of splenectomy

1. **Complications common to all operations:**
   Atelectasis, DVT or Reactionary hemorrhage.
2. **Specific:**
   a. Acute gastric dilatation (AGD)
   b. Portal vein thrombosis
   c. Pancreatic fistula may follow damage to tail of pancreas.
   d. Gastric fistula
   e. Hematemesis
   f. Subphrenic Hematoma or abscess
   g. Post splenectomy bacterial infections (Strept pneumonia. N. meningitides, H. influenza). Splenectomized children should receive anti-pneumococcal vaccine. Patients should commence Amoxil at first sign of febrile illness.

Causes of huge spleen

1. Bilharzial hepatosplenomegaly.

2. Gaucher's disease.

3. Malaria.

4. Chronic myeloid leukemia.

5. Thalassemia major.

Pancreas

Acute pancreatitis

It is a serious condition that leads to death in 10% of cases.

Etiology :

1. Bile duct stones (50%) :

(a) A stone may obstruct the pancreatic ducts.

(b) A stone in the ampulla of Vater may lead to regurgitation of bile into the pancreatic duct leading to activation of pancreatic enzymes.

(c) The passage of a stone through the ampulla may initiate an attack.

2. Excess alcohol intake (20%).

3. Trauma (5%) : may be accidental, operative or with ERCP.

4. Rare causes include : viral infection (mumps), hyperparathyroidism.

5. Idiopathic (20%).

Pathogenesis and morbid physiology :

1. Premature activation of the pancreatic digestive enzymes with autodigestion of the gland.

2. The severity of pancreatic inflammation ranges from mild edema, to hemorrhage **(hemorrhagic pancreatitis)**, and severe necrosis **(necrotizing pancreatitis).**

3. The peritoneal cavity contains blood stained exudate which may be localized beside the pancreas (subphrenic abscess or pancreatic-pseudo cyst).

4. Fat necrosis are opaque yellow-white areas on the peritoneal surface and omentum. Liberated pancreatic lipase splits fat into fatty acids and glycerol. Fatty acids combine with calcium to form calcium soap.

5. Protein breakdown products (vasoactive kinines and other mediators) absorbed lead to widespread [illegible] with hypotension and cyanosis.

Complications :

[A] Systemic complications :

1. Shock from :
   - Loss of plasma into the peritoneum and into the retroperitoneal space.
   - Loss of blood in hemorrhagic cases.
2. MOSF e.g. acute renal failure, acute respiratory distress syndrome.
3. Disseminated intravascular coagulopathy (DIC).
4. Paralytic ileus.
5. Tetany.

[B] Local complications : Pancreatic pseudocyst (discussed later) & pancreatic abscess.

Clinical features :

Symptoms :

1. History of the cause.
2. Severe upper abdominal pain commonly radiates to the back.
3. Vomiting.

General examination:

1. Evidence of hypovolemic shock and cyanosis.
2. The patient commonly sits leaning forwards as this position partially relieves the pain.

Local signs:

1. Epigastric tenderness and rigidity on firm deep palpation.
2. Discoloration of the skin in the loins **(Grey turner's sign)** and around the umbilicus **(Cullen' sign)** after two days.
3. Abdominal distension due to ileus.
4. Palpable mass in the epigastrium (pseudocyst).

Differential diagnosis:

Acute pancreatitis should be differentiated from other causes of **acute upper abdominal pain.**

Investigations :

[A] Laboratory :

1. Serum amylase : usually elevated to levels higher than 1000 IU/dL (normal value is 100-300 IU/dL) and remains elevated for 2-3 days.

- It is elevated in other conditions as perforated peptic ulcer, acute cholecystitis, intestinal obstruction, and acute mesenteric ischemia. The enzyme level in these cases usually does not exceed 500 IU/dl.

2. Arterial blood gases : to detect who will need mechanical ventilation.

3. Hypocalcaemia and hyperglycemia.

4. Leucocytosis.

5.urinary amylase: as the serum amylase is cleared out by the kidney, urinary amylase level can be used for diagnosis of patients who present after two days of the start of pain.

6.serum lipase: lipase has a slightly longer half-life. It is particularly useful in cases of delayed presentation. Elevated lipase levels are more specific to the pancreas than amylase levels.

The serum amylase level may be normal. The causes include: (A) **hyperlipidemia**, which interferes with chemical determination of amylase; (B) **increased urinary excretion of amylase**. And (C) **near complete destruction of pancreatic parenchyma**. On the other hand, the ***serum amylase level may be elevated in the absence of pancreatitis.***

[B] Radiological :

1. Plain X-ray of the abdomen may show :

- Dilated short segment of the small intestine (sentinel loop).
- Distension of the transverse colon and collapse of the descending colon **(Colon cut-off sign).**

2. Abdominal ultrasound : may show gall stones and enlargement of the pancreas.

3. CT scan : is very helpful. It shows enlargement of the pancreas and peri-pancreatic edema. Pancreatic necrosis is diagnosed when a big part of the parenchyma is not enhanced, in CT scan with I.V contrast.

4. Magnetic resonance cholangiopancreatography (MRCP): if choledocholithiasis is suspected.

N.B: ERCP is not recommended unless therapeutic procedure is planned

[C] ECG and cardiac enzymes: to exclude infarction.

Treatment

The treatment of acute pancreatitis is essentially conservative.

[A] Conservative treatment : Simply remembered as the "R" regimen :

1. Relief of pain : by pethidine. Morphine produces spasm of sphincter of Oddi and should not be used.

2. Replacement of the lost fluids : by crystalloids, and plasma. Blood is used in hemorrhagic cases.

3. Rest of the pancreas and bowel : no oral intake and nasogastric suction.

4. Respiratory support : by oxygen mask, or by mechanical ventilation.

5. Resistance of infection : by prophylactic antibiotics. **Carbapenemes** are the preferred antibiotics

6. Reassessment after improvement : by ERCP & extraction of any residual stone.

[B] Surgical treatment :

Indications :

1. Doubtful cases.
2. Local complications :
   - Pancreatic pseudocyst → internal drainage to the stomach or jejunal loop.
   - Abscess → external drainage.
3. In necrotizing pancreatitis : necrotic tissue is excised with drainage.

- **In biliary pancreatitis cholecystectomy is performed 6 weeks after the attack.**

Prognosis :

Ranson's criteria as shown below are used to identify patients at higher risk of mortality:

On admission		**Initial 48 hours**	
Age	>55 years	hematocrite decrease	> 10%
White cell count	>16000/uL	BUN elevation	> 5 mg/dl
Blood glucose	> 200 mg/dl	serum calcium	< 8 mg/dl
Serum LDH	>350IU/L	arterial PO2	< 60 mm Hg
AST	> 250 IU/L	base deficit	> 4 mEq/L

Chronic pancreatitis

Incidence :

Commoner in males than females, age above 50 years. Predisposing causes are chronic cholecystitis, alcoholism and in some cases hereditary pre-disposition is suspected.

Pathology :

- Slowly progressive destruction of the acini, with increasing fibrosis and later calcification.
- Pancreas becomes indurated, nodular and adherent to surrounding structures.

Clinical picture :

1. **Pain :** recurrent attacks of severe epigastric pain shooting to the back.
2. **Malabsorption disorder** with **steatorrhea, weight loss** and **vitamin deficiencies.**
3. **Obstructive jaundice** in some cases.
4. **Diabetes mellitus.**

Investigations :

1. **Stool :** shows increased fat excretion (steatorrhea).
2. **Blood and urinary amylase :** may be increased.
3. **Glucose tolerance test.**
4. **Plain X-ray :** may show pancreatic calculi.
5. **Ultrasound and CT scan.**
6. **ERCP :** multiple strictures in the pancreatic duct.

Treatment :

[A] Conservative treatment :

1. Control of pain.
2. Correction of the malabsorption by the intake of pancreatic enzyme tablets with meals.
3. Control of diabetes by insulin therapy.
4. Stop alcohol intake.

[B] Surgery :

Indications : persistent uncontrolled pain.

The type of operation may be :

1. Resection of a part of the pancreas.
2. Drainage of a dilated pancreatic duct e.g. pancreaticojejunostomy.

Annular pancreas

- It is a ring of pancreatic tissue that surrounds 2nd part of duodenum.
- May cause duodenal obstruction.
- **Vomiting** is the main symptom and jaundice may be present.
- Diagnosis reached by contrast radiological studies.
- Treatment: **Duodenojejunostomy**

Pancreatic cysts

Pancreatic pseudocyst

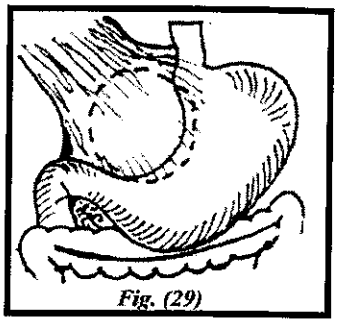


Fig. (29)

Definition :

This is a collection of pancreatic secretions and inflammatory exudates Within a lining of inflammatory tissue rather than epithelium, And that is why it is called a pseudocyst.

Etiology :

1. Acute pancreatitis. 2. Pancreatic trauma.

True cysts

Are either :

1. Acinar e.g. retention cyst, congenital cyst and cyst-adenoma.
2. Interacinar e.g. dermoid and hydatid cyst.

- **The commonest site of pseudocyst is behind and above the stomach.**

Clinical picture :Epigastric swelling placed centrally above the umbilicus and is fixed. It is tense cystic. It shows transmitted pulsations from the aorta which disappears in knee elbow position.

Investigations :

1. **Barium meal :** displacement and compression of stomach (in lateral view).
2. **CT scan** of upper abdomen (the most accurate investigation).
3. **Ultrasonography.**

Treatment :

- Small Pancreatic pseudocyst is painless and may be discovered by follow up sonography
- Pancreatic pseudocyst that is not resolved in 6 weeks requires drainage. It will be anastomosed to the stomach i.e. cystogastrostomy or to jejunum **i.e. cystojejunostomy.**.
- True cysts of the tail are treated by **distal pancreatectomy.**

Pancreatic neoplasms

Exocrine		Endocrine	
Benign	Adenoma	**Alpha cells**	Glucagonoma
Malignant	• Adenocarcinoma • Cystadenocarcinoma	**Beta cells**	Insulinoma
		Delta cells	Somatostatinoma
		Non beta cells	Gastrinoma (ZE syndrome)

Insulinoma

- **The tumor secretes insulin.**
- It is Usually benign and few cases are malignant. Some of them are part of MEN type one

Clinical picture : (Whipple's triad)

1. An attack of **hypoglycemia** occurs on fasting, with sweating, dizziness, blurring of vision and hunger.
2. During the attack blood sugar drops to below 50 mg%.
3. The symptoms are relieved by glucose.

- Hypoglycemia is accompanied by high insulin level in the blood confirms the diagnosis. and by selective venous sampling.
- Commonly found in the body and tail. Rarely multi-centric.
- Localization is difficult and may be accomplished by CT scan, selective angiography
- Intra-operative U/S is a very useful localization method.

Treatment :

1. Enucleation of the tumor. **OR** 2. Distal pancreatectomy.

Carcinoma of the pancreas

Incidence :

- **The tumor affects the head in 60% of cases :** 2/3 in the head proper and 1/3 is a periampullary carcinoma. The latter arises from the ampulla of Vater, the duodenal papillae, or the lower end of the common bile duct.
- **The body and tail in 40% of cases.**

P.F are chronic pancreatitis, alcohol abuse, high protein, high fat food and smoking.

Pathology : The tumor is either :

1. Spheroidal cell carcinoma, scirrhous or encephaloid.
2. Adenocarcinoma.

Spread :

Locally : To bile duct, duodenum and peritoneum.
Lymphatic : To regional lymph nodes then to the celiac axis and para-aortic nodes.
Blood stream : To the liver.

Clinical picture :

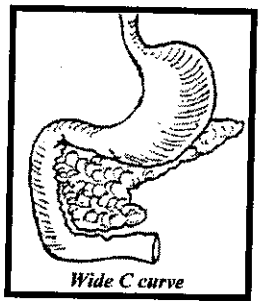
Sex : More in males. **Age :** usually above 50 years.

[A] Carcinoma of the head :

1. This tumor manifests relatively early by **obstructive jaundice** and pruritus.
2. The classic description is that of **painless and progressive** jaundice in an elderly male.
3. Sometimes it is painful.
4. The jaundice may temporarily lessen if an obstructing periampullary carcinoma sloughs.
5. The jaundice is usually deep olive green.
6. The liver is commonly enlarged because it is engorged with bile.
7. Gall bladder is palpably distended in half the cases.
8. Anorexia and loss of weight are marked.

[B] Carcinoma of the body and tail :

1. It is late to show its presence.
2. The manifestations are **nonspecific,** and include epigastric pain radiating to the back, anorexia, loss of weight, and weakness.
3. Migrating inflammation of the superficial veins (thrombophlebitis migrans) is known to accompany visceral malignancies.
4. Liver may be enlarged from metastases & ascites may be present.
5. Splenomegaly is present in 10% of cases due to splenic vein thrombosis.


Wide C curve

Differential diagnosis :

1. Calcular OJ (see OJ). 2. Other causes of jaundice. 3. Chronic pancreatitis.

Investigations :

1. **Barium meal :** shows
   a. widening of duodenal curve (in cancer head) or
   b. Inverted **3** (in periampullary carcinoma).
2. **Pancreatic insufficiency test.**
3. **Triphasic, spiral CT CT scan** is more sensitive than ultrasonography. it shows hypodense lesion. It is important to look for the invasion of sup. mesenteric vessels and the portal vein.
4. **Ultrasonography.**
5. **ERCP :** • Dilated duct with localized narrowing. • Can place a stent to relieve the obstruction.

- **CEA is a tumour marker for GIT carcinomas & is elevated in only 40-50% of patients.**
- **CA 19-9 is a method to confirm diagnosis and for follow-up.**
- **Biopsy is not needed if there are clinical evidence, elevated CA 19-9 & hypodense lesion on triphasic CT.**

Treatment :

- **Preoperative preparation :** see obstructive jaundice.

N.B. 5-flurourcil and Gemcitabine are recommended before and after operations.

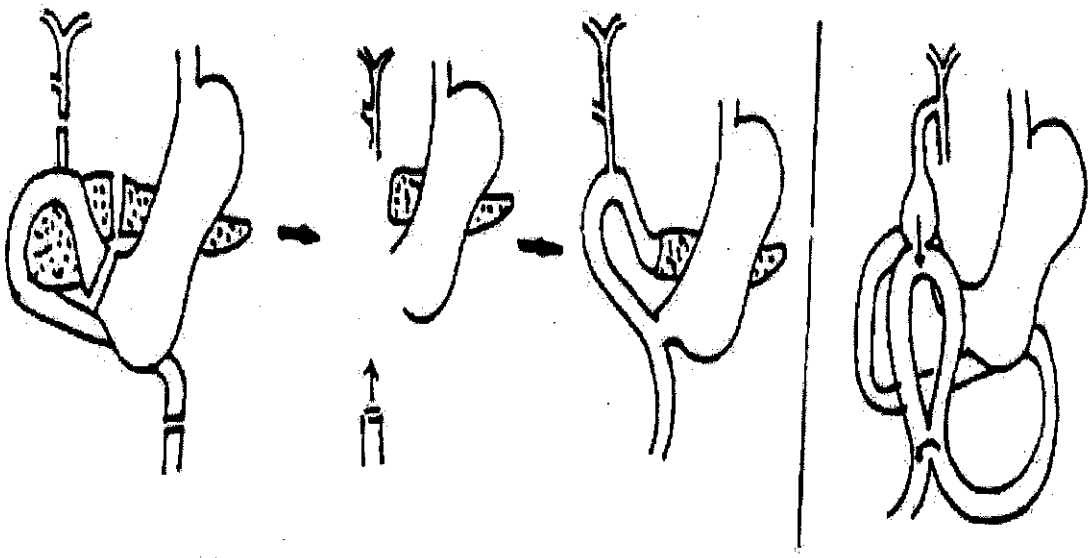
1. **Operable tumors of the body and tail :** are treated by removal of the **spleen** with the tail and body of pancreas.

2. **Operable tumors of the head :** are treated by pancreatico-duodenectomy **(Whipple operation)**
   - ➡ The head & neck of pancreas, whole duodenum, the antrum of stomach, G.B and CBD are removed.
   - ➡ Continuity is restored by anastomosis of pancreatic stump to the open end of jejunum, and the anastomosis of CBD and stomach to the side of the jejunum.

3. **Inoperable tumors of the head (the majority of cancers) :**
   (a) Chole**cysto**jejunostomy with enteroanstomosis. OR :
   (b) Chole**docho**jejunostomy to roux-en-Y. **Better**
   (c) Endoscopic stenting.

Prognosis :

- 5 year survival after resection is poor,
- It is slightly better with periampullary carcinoma (30%) than
- Carcinoma of the head proper (0-5%).



Pancreatico-duodenectomy

Cholecysto-jejunostomy & Jejuno-jejunostomy

Peritoneum

Peritoneum

Innervation :

- The parietal peritoneum is sensitive & is innervated by both somatic & visceral afferent nerves. The anterior parietal peritoneum is the most sensitive & any local insult to the parietal peritoneum leads to protective voluntary muscle guarding & then reflex muscular spasm. The visceral peritoneum receives innervation only from the autonomic nervous system & is relatively insensitive.
- When parietal peritoneal defects are created, healing occurs not from the edges but by metamorphosis of in-situ mesenchymal cells, large defects heal as rapidly as small defects.

Generalized septic peritonitis

Inflammation of the peritoneal cavity by pyogenic organisms.

Causes : Organisms reach the peritoneal cavity by :

1. Local spread from : (The commonest source of infection)
(a) Infected organs e.g. appendicitis, cholecystitis.
(b) Leaking organs e.g. perforated peptic ulcer, perforated typhoid ulcer, leaking anastomosis.

2. Direct entry : Through an operative or traumatic wound.

3. Blood spread : In cases of septicemia and pyemia (rare).

4. Primary peritonitis :

- **Definition :** Septic peritonitis with no apparent intraperitoneal pathology.
- **Incidence :** It usually affects female children ; infection is thought to reach the peritoneal cavity through the fallopian tubes.
- **Organisms :** Pneumococci and streptococci.

Fate :

The fate depends on the virulence of the organisms on one side and the efficiency of treatment and the body resistance on the other :

[1] Resolution : If the source of infection is controlled or removed.

[2] Localization (abscess formation) :

- If complete resolution fails, it occurs either :

1. Around the primary focus (e.g. in acute appendicitis).
2. In one of the anatomical compartments of the peritoneal cavity e.g. subphrenic, iliac or pelvic abscess.

[3] Flaring up :

- The factors which predispose to generalized peritonitis include :

1. **High virulence of the organisms.**
2. **Sudden perforation of a hollow viscus.**
3. **Persistent source of infection.**
4. **Stimulation of peristalsis** by eating, enemas or purgatives.
5. **Immunosuppression** as in diabetes mellitus.
6. **Generalized peritonitis** is likely to occur in children because the greater omentum is small in size and not well developed.

[4] Septicemia, multiple organ failure and death.

Clinical Picture :

- **Picture of the cause** e.g. acute appendicitis.
- **In late cases it may be difficult to tell the original cause.**

Symptoms :

1. Pain : Persistent, dull aching, and is increased by movements or coughing.
- The site of maximum pain is usually at the site of the original lesion.

2. Vomiting, abdominal distension, absolute constipation (due to paralytic ileus).
- If there is a pelvic collection the patient may have tenesmus or diarrhea.

Signs :

1. Inspection :
- Patient lying still without a movement and the abdomen is distended.

2. Palpation :
- Tenderness and rebound tenderness, rigidity at first localized to the primary site and later may be generalized.

3. Percussion :
- Shows shifting dullness.

4. Auscultation :
- Silent abdomen.

5. General examination :
- Rapid weak pulse, hypotension, high fever, sunken eyes, dry lips, cold clammy sweat, anxious look **(Facies Hippocratica).**

Investigations :

1. Blood picture : Leucocytosis (always present).

2. Plain X-ray abdomen :
- It demonstrates paralytic ileus with distension of the small and large bowel.
- It may determine the primary lesion, e.g. perforated peptic ulcer shows air under the diaphragm.

3. Abdominal ultrasound will reveal free fluid in the peritoneal cavity.

4. Peritoneal diagnostic aspiration.

Management :

[A] Pre-operative preparation :
- A naso-gastric tube, intravenous fluids & analgesics.
- Antibiotics (a combination of ampicillin, an aminoglycoside and metronidazole).

[B] Operation :
- Once the general condition of the patient is satisfactory, **An exploratory laparotomy** is performed.
- **Incision :** long longitudinal (midline or paramedian).
- **The pus is aspirated** (send for culture).
- **The primary lesion is dealt with.**
- **Peritoneal toilet** by copious irrigation with sterile saline and
- **Peritoneal drainage** and **drainage of the subcutaneous space.**

[C] Post-operative care :
- Admission in an I.C.U.

Localized septic peritonitis (Intraperitoneal abscesses)

Localization is favored by :

1. Anatomical arrangement of the peritoneal cavity into supra and infracolic and pelvic compartments.
2. The omentum (which is the policeman of the abdomen).
3. Loss of intestinal movement.
4. Fibrinous peritoneal exudate.
5. Rigidity of abdominal wall.

Iliac abscess

Etiology :

On the right	On either side	On the left side
Acute appendicitis. Perforated duodenal ulcer.	Spread from female genital organs. Secondary to generalized peritonitis.	Perforated diverticulitis. Perforation of sigmoid colon cancer.

Clinical picture :

Symptoms : Pain, swelling, hectic fever, vomiting and constipation.
Signs : Tenderness and rigidity over the site of the abscess. The overlying skin may show signs of inflammation.

Treatment :

- Drained through an extraperitoneal muscle cutting incision.
- Nowadays, it is possible to do percutaneous drainage of the abscess guided by ultrasound or CT scan.

Pelvic abscess

Definition :

A pelvic abscess is a collection of pus in the recto-vesical pouch or in Douglas pouch.

Causes :

1. Acute appendicitis.
2. Localization of resolving diffuse peritonitis.
3. Pelvic inflammatory disease in females.

Clinical picture :

1. Hectic fever and toxemia.
2. Deep pelvic pain.
3. Diarrhea and tenesmus are due to rectal irritation.
4. Burning micturition and frequency are due to bladder irritation.
5. Pelvic abscess may present suprapubically (mass, redness).
6. By digital examination there is fullness and tenderness on pressure in front of rectum.
7. If neglected, it bursts through the rectum or the vagina.

Treatment :

1. If the abscess is pointing in rectum, transrectal drainage is recommended.
2. If the abscess is pointing in vagina, the abscess is to be drained through the posterior fornix.
3. If the abscess is pointing suprapubically, suprapubic extraperitoneal drainage.

Subphrenic abscess

Anatomy of subphrenic spaces :

- The subphrenic region is considered as that portion of the abdominal cavity which extends from the diaphragm above to the transverse colon & mesocolon below.
- The region is divided by the liver into suprahepatic & infrahepatic compartments.
- The falciform ligament divides the suprahepatic compartments into right & left portions.
- **Taking these points into consideration the subphrenic spaces include the following :**

1. Right suprahepatic space.	2. Left suprahepatic space.
3. Right infrahepatic space **(Hepatorenal pouch of Morison).**	4. Left anterior infrahepatic space. 5. Left posterior infrahepatic space **(This is the lesser sac).**
6. Right extraperitoneal space.	7. Left extraperitoneal space.

Etiology of subphrenic abscess :

1. Residual pus collection following generalized peritonitis.
2. Inflamed or perforated viscera e.g. Appendix, gall bladder or peptic ulcer.
3. Lymphatic spread from chest infection.
4. Post-operative collection of infected bile after biliary surgery.

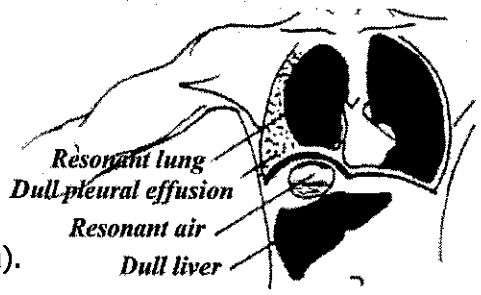
Clinical picture :

A subphrenic abscess should be suspected whenever a hectic pyrexia develops or persists after the treatment of any inflammatory lesion within the abdomen as stated,

"Pus some where, Pus no where else, Pus under the diaphragm"

[A] General :

1. Hectic fever.
2. Tachycardia.
3. Severe toxemia with anorexia, vomiting, sweating, wasting with rapid deterioration of the general condition.
4. Persistent hiccough.
5. Slight or absent pain but there is epigastric discomfort and pain referred to the shoulder of the affected side (phrenic nerve irritation).



[B] Local :

Inspection :

- Impaired chest movement on the affected side.
- Rarely bulging of the lower ribs or upper abdomen.

Palpation :

- Tenderness may be present over the lower ribs & intercostal spaces, or below the costal margin.
- There may be swelling and rigidity of the upper abdomen.
- Downward displacement of liver with upward displacement of the apex of the heart.

Percussion : If the abscess contains gas, 4 percussion zones may be elicited :

- Resonance of the lung.
- Dullness of the pleural effusion.
- Resonance of the gas in the abscess.
- Dullness of the liver or pus in the abscess.

Auscultation :

- Impaired air entry and crepitations over the lung base of the affected side may be detected.

Investigations :

1. White blood cell count shows leucocytosis and shift to the left are present.

2. Plain chest X-ray and screen :

- Thickened, elevated and fixed diaphragm **(tented diaphragm).**
- Obliteration of costophrenic space by a minimal pleural effusion may be seen.
- Gas under the diaphragm or air fluid level is sometimes seen when the cause is a perforated viscus, or when there is infection with gas forming organisms.

3. Ultrasound and CT scanning have proved very useful. They can localize the exact anatomical site and size of the abscess.

Treatment :

If conservative treatment fails, the abscess should be drained by an extra-serous approach to avoid contamination of pleural and peritoneal cavities :

1. The posterior extrapleural extraperitoneal approach (when located posteriorly) :

- The 12th rib is excised subperiosteally and a transverse incision is done in its bed in line with the first lumbar transverse process through the lowest fibers of the diaphragm.
- A finger is inserted and worked until the abscess bursts (above the kidney between the diaphragm and liver). Fibrous speta are broken to open all loculi.
- A drain is inserted.

2. The anterior extraperitoneal approach (when located anteriorly) :

- Small subcostal incision. All layers are divided but not including the peritoneum.
- A finger is inserted and worked until the abscess bursts, and a drain is left in place.

3. Recently a subphrenic abscess can be aspirated under ultrasound or CT guidance :

- A fine catheter is left in the abscess cavity to continue the drainage. Thick pus and a multilocular abscess are indications to abandon this technique in favor of open drainage.

Tuberculous peritonitis

Etiology :

The disease is always secondary to a tuberculous focus elsewhere that reaches the peritoneum through :

1. Direct spread e.g. tuberculous lymphadenitis, enteritis and salpingitis (the commonest cause).

2. Blood spread e.g. pulmonary tuberculosis.

3. Lymphatic spread e.g. from pleura or bowel.

Pathology : Five types are encountered :

1. The acute miliary type :

- The peritoneum is studded with tubercles.
- The exudate is straw colored.
- It resembles acute peritonitis and the diagnosis is frequently made during operation.

2. Caseous type (purulent form) :

- It affects young females as a complication of tuberculous salpingitis.
- The peritoneum is studded with tubercles and is thickened.
- Multiple collections of caseous material are present among the adherent bowel and omentum **(cold abscess form)** which may point to produce sinuses or fistulae.

3. Ascitic type (the commonest type) :

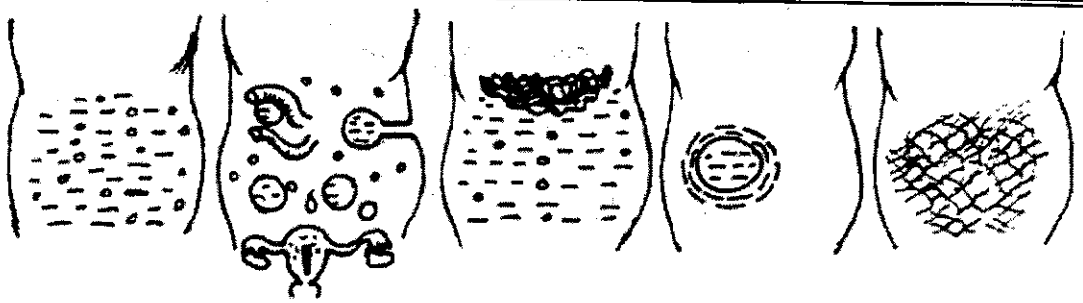
- The peritoneum is studded with tubercles.
- There is a copious amount of straw colored fluid.
- The greater omentum is thickened, fibrosed and frequently rolled up forming **a sausage shaped mass above the umbilicus.**

4. Encysted type (localized form of the ascitic type) :

- The fluid is encysted by fibrous adhesions and coils of bowel.
- It produces an intra-abdominal cyst which may be mistaken for ovarian and mesenteric cysts.

5. Adhesive type (fibrous or plastic form) :

- This type is characterized by extensive peritoneal adhesions which may lead to intestinal obstruction.



Acute milliary *Caseous* *Ascitis* *Encysted* *Adhesive*

Clinical Picture :

1. The disease usually affects children and young adults of both sexes.
2. There are recurrent attacks of abdominal pain, vomiting and distension.
3. Tuberculous toxemia with night fever, night sweats, anorexia and wasting is present.
4. The abdomen is felt doughy with multiple palpable swellings which may be lymph nodes, omentum and tuberculomas. Ascites is a common finding.
5. Tenderness and guarding may be present.
6. A sausage shaped mass above the umbilicus may be felt **(rolled omentum).**
7. Per-vaginal examination may reveal a tubo-ovarian mass.

Investigations :

1. **Blood picture** may reveal anemia and lymphocytosis.
2. **ESR** is raised.
3. **Tuberculin test** is highly positive.
4. **Abdominal ultrasound.**
5. **Abdominal tapping.**
6. **Laparoscopy.**
7. **Exploration.** In many patients the diagnosis is only reached after exploration.

Treatment :

- At least 2 anti-tuberculous drugs e.g. (isonicotinic acid hydrazide and rifampicin) for at least 1 year.
- Operation is indicated in a few cases e.g. intestinal obstruction to relieve a band of adhesion or to perform a lateral anastomosis around an obstructed area.

Differential Diagnosis of Ascites

Items	Cirrhotic	Malignant	Tuberculous
Age	Any	Elderly but may be young in ovarian cancer	Usually young
History	Jaundice or Hematemesis	Short history may symptoms of the 1ry	Toxemia
G exam	Liver insufficiency	Distant metastases	Toxemia
Abd exam	HSM may be present	Multiple Hard Abd masses may be palpable	Doughy mass may be palpable
LFT	Poor	Normal	Normal
U/S	Liver cirrhosis / Splenomegaly	Abd masses may be detected	Abd masses may be detected
Tapping	Transudate	Exudate & cytology may show malignant cells	Straw colored exudates & C/S may reveal TB bacilli
Treatment	Medical Peritoneo-venous shunt	Tapping Radioactive gold	Anti-tuberculous
Prognosis	Bad	Very bad	Good

The mesentery

Tabes mesenterica (Tuberculous mesenteric adenitis)

This is a childhood type of intestinal tuberculosis due to ingestion of contaminated milk. **It consists of :**
1. A small focus in distal ileum (Peyer's patches).
2. Huge enlargement of mesenteric nodes.

Fate :

Resolution	Activation
• Healing by fibrosis and calcification. • Later adhesions and kinking may produce intestinal obstruction.	• Caseation produces a mesenteric cold abscess. • Rupture in peritoneal cavity produces TB peritonitis. • Spread to thoracic duct may cause miliary tuberculosis.

Clinical features (during activity) :

Symptoms :
1. Tuberculous toxemia. 2. Abdominal pain with vomiting and diarrhea.

Signs :
1. Tenderness below and to the right of umbilicus. 2. Firm irregular tender masses may be felt (nodes).
3. In acute cases : severe tenderness, rebound tenderness and rigidity, in the right iliac fossa simulating appendicitis, or along the line of the mesentery.

Investigations :
1. Leucocytic count is normal.
2. Tuberculin test. If the test is negative, tuberculosis is excluded.
3. X-ray of the abdomen may show calcified nodes as mottled shadows arranged along the line of the small intestinal mesentery.

Treatment :
Antituberculous treatment is given for active cases. Calcified uncomplicated nodes need no treatment.

Mesenteric cyst

Definition : Fluid collection between the 2 layers of mesentery of the small bowel. **It may be :**

(A) False mesenteric cyst (without epithelial lining)	(B) True mesenteric cyst (with epithelial lining)
1. Blood cyst. 2. Tuberculous mesenteric cold abscess.	1. Chylolymphatic cyst. 2. Enterogenous cyst. 3. Teratomatous dermoid cyst. 4. Hydatid cyst.

Items	Chylolymphatic cyst	Enterogenous cyst
Origin	Retention cyst due to obstructed lymphatic drainage.	From sequestrated intestinal epithelium. **OR:** From duplicated intestine.
Structure	Thin walled, lined with endothelium & contains lymph **(chyle).**	Thick walled, lined with intestinal mucosa & contains mucous.
Blood Supply	Its blood supply is separate from that of the related loop of intestine.	Its blood supply is derived from the same vessels of the related loop of intestine.
Treatment	Enucleation leaving the related intestine intact.	Excision with resection anastomosis of the related loop of intestine.

Tillaux triad

1. The cyst is near the umbilicus.
2. It moves across but not along the root of mesentery.
3. The cyst is dull on percussion but there is a zone of resonance around it and a band of resonance over it due to the presence loops surrounding and infront of it.

Peritoneal Tumors

☒ **Carcinoma peritonii**

1. It is a terminal event in many cases of abdominal malignancy, caused by implantation of secondaries on the peritonium.
2. Malignant ascitis
3. Peritoneal metastasis (Discrete nodules, Plaques variable in size &color, Diffuse adhesions which may be complicated with frozen pelvis)
4. Treated by instillation of radioactive gold intra-peritoneally after paracentesis.

☒ **Pseudomyxoma peritonii**

a. It is due to rupture pseudo-mucinous cyst of the ovary and rupture of mucocele or mucoid carcinoma of appendix
b. The abdomen is filled with yellow jelly-like material (Pseudomucin)
c. By abdominal examination, the abdomen is distended without shifting dullness. Multiple firm masses are palpable.
d. At laparotomy
   - The masses of jelly like material are scooped out
   - The primary focus is removed
e. Unfortunately, it is liable to multiple recurrences

☒ **Mesothelioma**

a. It is a primary neoplasm of peritoneum.
b. It is suspected to be caused by exposure to asbestos
c. It is either diffuse causing ascites, or localized and bulky in which case it presents as an abdominal mass.

Retropertoneal tumors

1. **Renal and adrenal tumours**
2. **Retroperitoneal sarcoma:**
   - This commonly presents by an abdominal mass, which is fixed and usually hard. The patient may complain of vague abdominal pain.
   - The size of the lesion outmatches the symptoms caused by it.
   - If the lesion infiltrates the colon, the patient may complain of chronic large bowel obstruction. If the ureter is obstructed by the tumor, the patient may complain of renal pain.
   - Ultrasound and CT scan are very useful for the diagnosis.
   - Treatment: The patient is explored for the possibility of resection.
   - Unfortunately, most of the cases are un-resectable and all what can be done is to take a biopsy.
   - Radiotherapy may offer some palliation.
3. **Retroperitoneal LN enlargement**: is commonly due to lymphoma.
4. **Retroperitoneal lipoma**:
   - It is more in females , grows slowly and may attain large size.
   - It is liable to myxoematous and malignant transformation.

Torsion Omentum

1. It is due to adhesions of the omentum to a hernia or an old focus of infection.
2. It presents with sudden abdominal pain with rigid tender abdomen. The twisted omentum may be palpable.
3. Treated by excision of the strangulated omentum.

Appendix

Acute appendicitis

It is the commonest cause of acute abdomen with serious complication which may occur in neglected cases. In western countries 16% of the population undergoes appendicectomy.

Predisposing factors :

1. Age :

- It is rare in infants (short appendix with wide base).
- The incidence increases with age & becomes maximum at 20-30.
- In elderly it becomes rare again (appendix atrophies).
- Although appendicitis is rare in extreme of age, it is most serious.

2. Race and diet : Appendicitis is commoner in more civilized cases.

3. Social status : It is commoner in higher social classes.

4. Familial susceptibility : May be due to hereditary abnormality in appendix position e.g. all family has long retrocecal appendix.

5. Obstructed appendix : By fecolith, stricture, kinking, ascaris, oxyuris, fruit seeds, foreign bodies.

6. Septic focus : from which organisms are carried to the lymphoid follicles of the appendix e.g. upper respiratory tract infection. It may be the cause of what is called epidemic attacks of acute appendicitis.

Source of infection and type of organism :

1. Direct from the lumen, the organism may be : E. Coli OR Strept fecalis.
2. Blood borne (rare), the organism is usually : Streptococci.

Pathology :

Non-obstructive (Slowly developing inflammation)	Obstructive (Rapidly developing inflammation)
Less common (1/3 of cases).	More common (2/3 of cases).
Less serious (free drainage).	More serious as pus collects under tension (empyema of appendix).
Infection starts in mucosa or lymph follicles.	Infection starts in mucosa or lymph follicles.
Fate : Spontaneous resolution with residual fibrosis may occur.	**Fate :** Appendix is tense with pus.
Slow spread of infection to all coats.	Infection spreads rapidly to all coats.
Blood supply to tip is strangulated **slowly** with gangrene and perforation.	Blood supply to tip is strangulated **rapidly** with gangrene and perforation.
Resulting peritonitis is **localized** as perforation occurs **slowly** (24 hours) giving chance for localization.	Resulting peritonitis is **generalized** as perforation occurs **rapidly** (12 hours) before localization.

- **Both obstructive & non obstructive may be catarrhal, suppurative or gangrenous.**

N.B.:

The appendicular arteries run in the mesoappendix, but they are closely applied to the wall distally, therefore, with acute appendicitis, thrombosis with consequent gangrene is likely to affect the distal part of the appendix.

Complications of acute appendicitis :

[1] Appendicular mass	[2] Appendicular abscess
Occurs within 2-3 days in neglected cases with good resistance. It consists of inflamed or perforated appendix surrounded by edematous cecum, loops of intestine and greater omentum.	**In right iliac fossa or pelvis from :** 1. Localized peritonitis if perforation is delayed (after 24 hours); as in non-obstructive type. **OR :** 2. Suppurating appendicular mass.
Signs : Firm tender mass either in Rt iliac fossa with overlying rigidity, or in pelvis, felt by PR.	**Signs :** Cystic tender mass, rigidity and hectic fever.
Fate : 1. Resolution by antibiotics. 2. Appendix abscess. 3. Generalized peritonitis.	**Fate :** 1. Resolution if drained. 2. Pointing on skin. 3. Peritonitis.

[3] Generalized peritonitis :

- From gangrene and perforation (usually at tip). If perforation occurs early (12-18 hours), as in obstructive appendicitis, peritonitis is generalized.
- Acute appendicitis is more liable to cause generalized peritonitis in children, elderly persons, during pregnancy, diabetics and in patients who had purgatives or enema.

[4] Subphrenic abscess : Follows peritonitis if pus trickles up along paracolic gutter in lying down patients.

[5] Portal pyemia : From portal spread of infection (The most serious position is the preileal position as it carries the possibility of thrombophlebitis of the ileo-colic vein is the portal pyemia).

[6] Perforation of mucocele : resulting in pseudomyxoma peritonii.

[7] Recurrent subacute appendicitis :

- It is a cause of recurrent attacks of abdominal pain and dyspepsia.
- It is advisable to exclude other causes of chronic pain before removing the appendix.
- **Investigations :** Barium follow through.
- **Treatment of chronic appendicitis :** Appendicectomy.

Clinical features :

- **Proper history taking and physical examination are the corner stone of diagnosis.**

Symptoms :

1. Acute pain that shifts is the presenting symptom. Classically it starts in the periumbilical region and is ill localized. Distension of the appendix produces this **(visceral pain)** that is felt in this area because the appendix is a part of the midgut. Within 6 - 10 hours when the inflammation proceeds to irritate the parietal peritoneum, the pain shifts to the right iliac fossa and becomes sharper and more localized **(somatic pain).** The pain is aggravated by movement or cough.

2. Anorexia and nausea are nearly always present. Vomiting occurs in about three fourths of the patients, is not equally repeated, but is always preceded by the pain.

3. Constipation is usual. Relation of constipation and acute appendicitis :

- Etiology.
- Result : due to localization in the right iliac fossa.

Physical examination :

[1] Temperature and pulse are only slightly elevated early in the disease. Higher elevations indicate a complication.

[2] If the patient is asked to cough, the pain becomes sharp and well localized to the site of the appendix **(cough tenderness).**

[3] Localized tenderness (and probably rebound tenderness) is present in the right iliac fossa, usually, but not always, over the **McBurney's point** (most important sign to diagnose appendicitis).

- It is the site of the base of the appendix.
- Not tender in displaced cecum.

[4] Pelvic examination either vaginal or rectal is essential to exclude gynecological problems.

- **It also reveals tenderness or mass in case of pelvic appendicitis.**

[5] Special signs, if present, assist in the diagnosis :

1. Rovsing's sign is pain felt in the right lower quadrant on deep palpation of the left lower quadrant.

2. Psoas sign is pain on extension of the right thigh. It is caused by psoas spasm as a result of irritation by retrocecal appendix.

3. Obturator sign is pain on internal rotation of the flexed right thigh that is caused by irritation of the obturator internus by pelvic appendicitis.

4. Hyperesthesia in Sherren's triangle (between umbilicus, symphysis pubis and right anterior superior iliac supine). It disappears after perforation.

[6] Clinical picture of complications :

1. Appendicular mass	2. Appendicular abscess
History of 2-3 days.	History of few days. (localized peritonitis or suppurating mass).
Temperature over 38^{0} C.	Hectic fever.
Firm tender mass in right iliac fossa with rigidity or in pelvis felt by PR.	**Cystic tender mass** in right iliac fossa with rigidity or in pelvis felt by PR.

3. Picture of peritonitis :

- Generalized rigidity, restricted abdominal wall movement with respiration.
- Deterioration of general condition

4. Portal pyemia :

- Chills & high fever.
- Low grade jaundice.
- Hepatic abscesses.

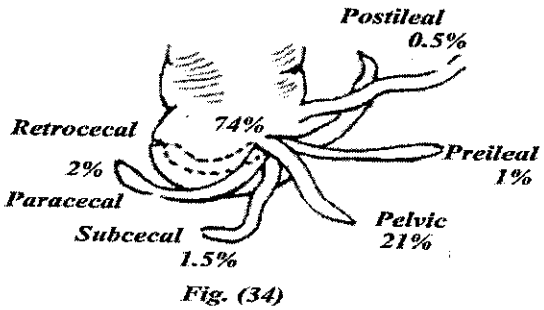


Fig. (34)

N.B.:

The commonest position of the appendix is retrocecal (74 %).
The second common is the pelvic position (21 %).
Less common are the paracecal (2 %), the subcecal (1.5 %), preileal (1 %).
The least of all positions is the post-ileal (0.5 %) appendix.

Atypical clinical features :

1. Pelvic appendix : May induce tenesmus from rectal irritation, and dysuria from bladder. Irritation. Abdominal signs are minimal and tenderness is elicited on pelvic examination.

2. Subhepatic appendix : Pain and tenderness are higher than the McBurney's point.

3. Long retrocecal appendix : Tenderness is felt in the right loin. Abdominal examination reveals minimal tenderness or guarding.

4. Retro-ileal appendix : Diarrhea may occur when the ileum is irritated.

5. Appendicitis in infants and young children : Is more serious. Below the age of 3 years, 80% of patients will have perforated appendicitis due to the following reasons :

(a) Difficulty of examination of an infant or young child.
(b) The greater omentum is not well developed and so localization is difficult.
(c) There is prominent vomiting and the case may wrongly be diagnosed as gastritis or enteritis.

6. Acute appendicitis in the elderly is serious as :

(a) It produces little tenderness and rigidity.
(b) Perforation is relatively common due to atherosclerosis.
(c) The natural weakness of the immune system.

7. Appendicitis with pregnancy is serous as :

(a) The site of pain is displaced upwards as pregnancy progresses.
(b) The localization by the omentum is less efficient.
(c) The condition is often misdiagnosed as pyelitis.

Investigations :

1. Leucocytic count : A moderate leucocytosis (10.000 - 16.000/uL) with shift to the left(present in 80-90% of cases). However a normal white cell count does not rule out appendicitis.

2. Urine analysis: may show few red blood cells. The presence of a large number of RBC's, or of pus cells raises the suspicion of a ureteric calculus or of urinary tract infection respectively.

3. Serum amylase and other investigations to exclude D.D.

4. Ultrasound examination:

a) may diagnose appendicitis:
- The appendix appears distended and noncompressible
- A fecolith may also be detected.
- In children: the inflamed appendix is usually > 10 mm in diameter.

b) It is more important in exclusion of other diseases e.g. females with suspected gynecological problems and urological problems.

5. Laparoscopy : Diagnostic and therapeutic by laparoscopic surgery. Tubal pregnancy, ovarian cysts, and acute appendicitis, for example, can be managed laparoscopically.

Differential diagnosis :

I- Other causes of acute abdomen:

[A] Thoracic problems: *Right sided pneumonia or right basal pleurisy.*

[B] Upper abdominal problems :

1. Perforated peptic ulcer (discuss briefly).
2. Acute cholecystitis and biliary colic.
3. Acute pancreatitis.
4. Cyclic vomiting of children. Temperature is normal, Rigidity never occurs & Leucocytic count is normal.
5. Viral hepatitis,amoebic liver abscess and pyogenic liver abscess.
6. Leaking abdominal aortic aneurysm.
7. Intestinal obstruction,peritonitis,mesenteric vascular occlusion

[C] Lower abdominal problems :

1. Non-specific mesenteric lymphadenitis. The tenderness shifts with changing of position. It is difficult to be sure about the diagnosis.
2. Iliac adenitis.The site of maximum tenderness is much lower near the iliac vessels.
3. Cancer of the cecum. This is more likely to be misdiagnosed as appendicular mass.
4. Crohn's disease.
5. Meckel's diverticulitis. Usually not diagnosed except after exploration.
6. Intestinal obstruction.
7. Peritonitis.
8. Typhoid fever.
9. Mesenteric vascular occlusion.
10. Acute diverticulitis.

[D] Pelvic problems :

1. ***Gynecological and obstetric problems:***
   - Ruptured graffian follicle (mittleschmerz) may produce mid-cycle ovulation pain.
   - Ruptured ectopic pregnancy.
   - Tubal abortion.
   - Twisted ovarian cyst.
   - Pelvic inflammatory disease (PID) e.g. salpingitis, and tubo-ovarian abscess.
2. Proctitis ,cystitis, prostatitis.

[E] Urological problems :

1. Right Renal ache or ureteric colic.
2. Right pyelonephritis :

[F] Neurological problems & medical causes of acute abdomen :

1. Myocardial infarction.
2. Uremia (hyponatremia causes cramps).
3. Diabetic ketoacidosis.
4. Hemolytic crisis.
5. Familial Mediterranean fever.
6. Acute hepatitis.
7. Active peptic ulcer.
8. Severe gastroduodenitis.
9. Enterocolitis.
10. Basal pneumonia & pleurisy.
11. Herpes zoster.

The commonest D.D. of acute appendicitis

1. Right tubo-ovarian causes.
2. Right ureteric stone.
3. Meckel's diverticulitis.
4. Gastroenteritis.
5. Mesenteric adenitis.

N.B: causes of abdominal and back pains:

- Penetrating duodenal ulcer
- Acute cholecystitis and biliary colic.
- Acute pancreatitis.
- Leaking abdominal aortic aneurysm.
- Renal ache or ureteric colic.
- Spine disease with radicular pain

II- Differential diagnosis of appendicular mass:

- **The commonest cause of mass in the right iliac fossa is appendicular mass.**

1. Appendicular abscess.
2. Carcinoma of the cecum. The mass is usually hard and not tender.
3. Crohn's disease.
4. Hyperplastic ileocecal tuberculosis.
5. Right iliac lymphadenitis.
6. Actinomycosis.

Treatment :

[A] Non-complicated acute appendicitis :

Treatment is appendicectomy (either open or laparoscopic). The operation should not be delayed especially in children, elderly persons, pregnant females and diabetic patients.

[B] Complicated cases :

I- Appendicular mass : Urgent appendicectomy is not recommended because :

1. An appendicular mass represents success of the body to isolate the danger.
2. Appendicectomy is difficult and carries the hazard of injuring the intestine

Initial conservative treatment (Ochsner-sherren treatment) :

1. Rest in bed.
2. Intravenous fluids are administered until the nausea subsides.
3. Liquid diet for the first few days, then gradual introduction of solid meals.
4. Antibiotics : A combination of ampicillin, aminoglycoside and metronidazole.
5. Observations are made for the temperature, pulse, pain, vomiting, degree of tenderness and rigidity and the size of the mass.

- In 80-90 % of patients the appendicular mass will resolve under conservative treatment.
- The patient is discharged & scheduled for interval appendicectomy after 3 months.

II- Appendicular abscess :

- The pus should be drained by open surgery, or by ultrasound-guided percutaneous aspiration.

Open drainage :

1. If the abscess is in the right iliac fossa : the operation is done through an iliac muscle cutting incision to reach the abscess cavity through an extraperitoneal approach.

- The pus drained, and a rubber drain is left.
- No attempt is made to remove the appendix.

2. A pelvic abscess : Drained transanally through the anterior wall of the rectum.

- The patient is then scheduled for **"interval appendicectomy"** after three to six months.

III- Perforated appendicitis with generalized peritonitis : Urgent surgery is required. The perforated appendix should be removed and peritoneal toilet is performed.

IV- Portal pyemia : Urgent appendicectomy + Ligation of ileo-colic vein.

V- Recurrent subacute appendicitis : Appendicectomy either open or laparoscopic.

Neoplasms of the appendix

- **Carcinoid tumor (Argentaffinoma) :** This is the commonest tumor of the appendix. For the tumor is less than 2 cm, and is still confined to the appendix, appendicectomy is a sufficient treatment. For larger lesions, right hemicolectomy is required.
- **Adenocarcinoma :** The treatment is right hemicolectomy.

Small & Large Intestine

Principles of colon surgery

Compared to the small bowel, colonic anastomosis is more liable to leakage and peritonitis.

The factors of difference are :

1. The highly infective content of both aerobic and anaerobic organisms.
2. Constant gaseous distention.
3. Incomplete serous coat.
4. Peculiar blood supply where the terminal arteries poorly connect with each other.
5. Incomplete longitudinal muscle tone.

Preoperative preparation

I- Patient counseling : particularly with those who are likely to have an intestinal stoma (colostomy or ileostomy).

II- Improving nutritional status of the patient.

III- Bowel preparation :

[A] Mechanical preparation :

1. Non residue diet for 4 days before surgery.
2. Enemas and mild laxatives are given for 2-3 days before the operation.

Rapid preparation is and alternative that can be performed one day before surgery by :

1. Whole gut irrigation using 2-4 L/hour of saline passed via a nasogastric tube until the patient passes clear fluid per rectum.
   - Contraindications : (a) Cardiac or renal disease. (b) Partially obstructed colon.
2. One liter of mannitol administered orally or by a naso-gastric tube.

[B] Chemical preparation :

A combination of neomycin and metronidazole (Flagyl) for two days will cover the Gram negative bacilli and the anaerobes.

IV- Perioperative antibiotic prophylaxis.

Operative procedure

I- Elective (resection & anastomosis) :

[A] Resection :

The extent of resection is governed by the arterial blood supply and by the disease process. In radical surgery for malignant tumors, it is essential to remove the draining lymphatics. Division of the peritoneal attachments allows adequate mobilization of the bowel on its mesentery.

[B] Anastomosis :

Bowel ends should be adequately vascularized, and should be sutured without tension. An intestinal anastomosis can be done by one of two methods :

1. Two layers of interrupted sutures : The 1st layer includes the whole wall thickness, while the 2nd incorporates the serosa and muscle only, (Some surgeons prefer a single layer of interrupted sutures).

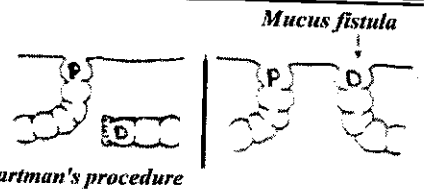
2. Mechanical staplers : A doubtful anastomosis in the distal colon or in the rectum can be covered by a temporary proximal loop colostomy in the transverse colon. The colostomy is closed after the anastomosis has healed.

II- Emergency surgery :

- In critically ill obstructed cases, a temporary proximal colostomy to decompress the colon is done, postponing resection to a later date.
- If the patient's condition allows, resection of the diseased colon should be carried out. Further management depends on the site of the resected part.

[A] The right colon :

The ileum is anastomosed to the transverse colon i.e. ileotransverse anastomosis.



Hartman's procedure

[B] Other parts of the colon : The options are :

1. The proximal colon end is opened to the skin as a temporary colostomy and the distal end is closed by sutures and replaced in the abdomen **(Hartmann's procedure).**
2. Both ends are opened to the skin, the proximal one as a temporary colostomy, and the distal one as a mucus fistula.

- **In either case a second operation is needed to restore bowel continuity within few weeks.**
- **This second elective operation should be preceded by proper bowel preparation.**

Intestinal trauma

Colon injuries are more dangerous than those of the small intestine (see before).

Etiology : As general.

Sequelae :

1. Peritonitis occurs due to the escape of intestinal contents into the peritoneum.
2. Internal hemorrhage.
3. Hypovolemic and septic shock.
4. Paralytic ileus.
5. Injuries may heal by strictures and massive adhesions presenting later by intestinal obstruction.

Treatment :

1. ABCDE.
2. Antibiotics that cover Gram-negative bacilli and anaerobes.
3. Tetanus toxoid.

Operation :

- Full abdominal exploration.
- Bleeding should be urgently stopped.

I- Small intestinal and right colon injuries :

1. Sharp injuries : Sutured in two layers.

2. Lacerated injuries : Trimming of the edges, and suturing.

3. Intestinal resection & 1ry anastomosis : Indications :

(a) Ischemic or gangrenous segments.
(b) Extensively contused segments.
(c) Multiple tears that are very close to each other.

II- Transverse and left colon injuries :

1. Localized injuries are exteriorized; it thus acts as a colostomy.

2. If the injured part cannot be exteriorized, the tear is sutured and is protected by a proximal colostomy.

3. In cases where resection of a segment is done, the proximal end of the colon is brought out as a terminal colostomy. The distal end is either brought out as a mucus fistula or is closed **(Hartmann's procedure).**

- **Elective restoration of bowel continuity is done after three weeks :**

A good nutritional status allows resection of the diseased bowel segment and anastomosis in one stage.

III- Internal fistula : (see later).

Intestinal fistulae

Definition :

A fistula is an abnormal communication between two epithelialized surfaces.

Pathology :

Classification :

1. Internal : when they connect the intestine to hollow viscera (another part of the gut, urinary tract or vagina).
2. External :
   (a) Low output fistula : discharge less than 500 ml/day.
   (b) High output fistula.

Complications :

1. Metabolic effects : malnutrition, dehydration, hypoalbuminemia, acid-base & electrolyte disturbances.
2. Sepsis : producing an intraperitoneal abscess.
3. Irritation : of the skin.

Etiology :

[A] After an abdominal operation (80%) : due to unrecognized intestinal injury, or a failure of an intestinal anastomosis.

[B] Other causes :

1. Congenital anomalies as a patent vitello-intestinal duct.
2. Abdominal trauma.
3. Inflammatory conditions as colonic diverticulitis & Crohn's disease.
4. Malignant tumors.

Management :

- **The great majority of intestinal fistulae heal spontaneously if sepsis is eradicated, the nutritional status is maintained and distal obstruction, if present, is relieved.**

1. Resuscitation and skin protection.

2. Nutritional support.

3. Investigations and eradication of sepsis :

- Clinical assessment.
- Fistulography and barium meal follow-through.
- Ultrasound or CT scan of the abdomen for detection of abscess cavities.

4. Suppression of intestinal secretions by Somatostatin 0.1 mg S.C. every 8 hrs until fistula closes or for 2-3 weeks.

5. Definitive treatment :

[A] External fistula : (spontaneous closure is common)

- Surgical intervention is indicated is for :

1. Cases that show no improvement over 4-6 weeks.
2. Distal obstruction.
3. Active disease at the fistula site e.g. Crohn's disease or malignancy.

- A good nutritional status allows resection of diseased bowel segment and anastomosis in one stage.

[B] Internal fistula :

- Spontaneous closure is rare to occur, and many internal fistulae don't require correction.
- Surgical correction is indicated for:

1. Patients with nutritional problems where a fistula bypasses a long segment of intestine producing malabsorption.
2. Internal fistula that connect the intestine with the urinary tract.

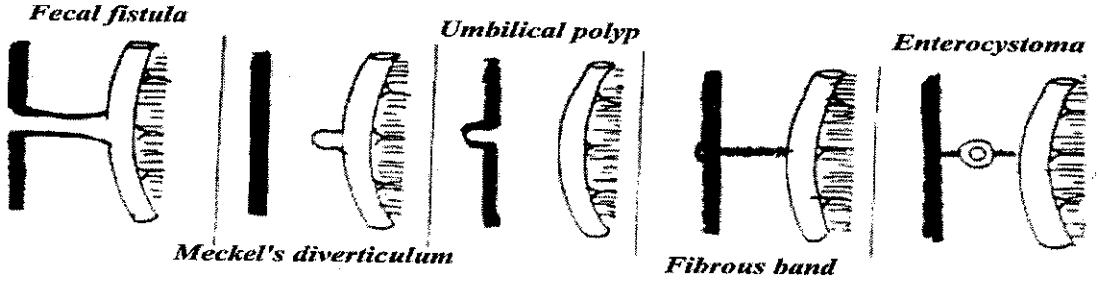
Intestinal diverticulae
Meckel's Diverticulum

Etiology :

- The most prevalent congenital anomaly of the gastrointestinal tract.
- It occurs in 2% of the population, in the ileum 2 feet from the cecum, and averages 2 inches in length.
- It is caused by persistent patency of the proximal part of the vitello-intestinal (omphalomesenteric) duct of the embryo.

- **The abnormal persistence of the duct may produce other anomalies as well :**

1. A fecal fistula connecting the ileum with the umbilicus, caused by persistent patency of the whole vitello-intestinal duct.
2. A persistent umbilical extremity of the duct is everted producing an umbilical polyp.
3. A fibrous band remaining after obliteration of the duct may cause volvulus.
4. An enterocyst that results from the persistent patency of the middle part of the duct (Enterocystoma).



Pathology :

- Meckel's diverticulum is **a true diverticulum (incorporates all layers of the bowel wall).**
- It lies on the antimesenteric border, and has its separate blood supply via a branch of the superior mesenteric artery.
- It may contain ectopic gastric mucosa and sometimes pancreatic cells.

Complications : Occur in about 4% of cases :

1. Intestinal obstruction : It may be due to :

- Intussusception.
- Pressure on an intestinal loop by a fibrous band that may attach the diverticulum to the umbilicus, the band may also allow rotation of the ileum around its axis **(volvulus).**

2. Peptic ulceration and bleeding : Caused by the ectopic gastric mucosa. The ulceration occurs in the intestinal mucosa adjacent to the base of the diverticulum.

3. Acute diverticulitis : Is similar to acute appendicitis. Clinically it is impossible to differentiate the two conditions. Finding a normal appendix at operation, the surgeon should look for a Meckel's diverticulum.

4. Incarceration in an inguinal or a femoral hernia is called Litter's hernia.

Clinical features :

A Meckel's diverticulum may be accidentally discovered at operation for another pathology or it may present by one of its complications.

Treatment :

[A] Symptomatic cases : Resection is indicated.

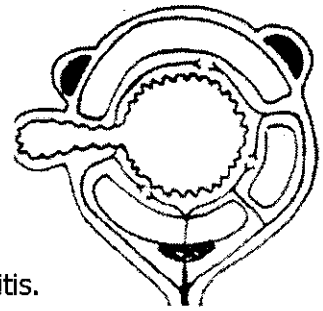
[B] Accidentally discovered at laparotomy :

Resection is indicated in children and young adults and in those with an attached band.

In patients over 40 years : It is better not to resect it & give the patient card that he has Meckel's diverticulum.

Diverticular disease of the colon

This disease represents pulsion diverticulae of the colonic mucosa through the circular muscle layer at the points of entry of the blood vessels between the taenia coli.



Etiology :

- Chronic constipation and increased intraluminal colonic pressure.
- Increased muscle spasm and segmentation may play a role.

Pathology :

1. Stage of non complicated diverticular disease or diverticulosis.
2. Acute diverticulitis.
3. Chronic diverticulitis is the end result of recurrent attacks of acute diverticulitis.

Site :

- The sigmoid colon is the commonest site affected, but any area of the colon may be involved.
- The rectum is not affected by the disease.

Complications :

1. **Recurrent attacks of acute inflammation** (acute diverticulitis).
2. **Perforation** which may lead to localized or generalized peritonitis.
3. **Bleeding** due to erosion of a blood vessel (may be massive).
4. **Chronic diverticulitis** may lead to a **stricture** with large bowel obstruction.
5. **Fistula formation :** A colo-vesical, colo-vaginal or colo-intestinal fistula may develop.

Clinical picture :

- Diverticular disease is uncommon before the age of 40 then the affection increases with age.

1. Stage of diverticulosis : The patient may be asymptomatic or complains of recurrent attacks of lower abdominal pain, distention and flatulence.

2. Acute diverticulitis : mimics the clinical picture of appendicitis but on the left side. It may be complicated by a pericolic abscess or by generalized peritonitis.

3. Chronic diverticulitis : Long history of recurrent attacks of pain with the passage of blood and mucous per rectum. On palpation there is a tender mass in the left iliac region. This mass has to be differentiated from carcinoma.

4. Bleeding : It is one of the two commonest causes of massive bleeding per rectum (Besides angiodysplasia of the colon). Angiography is the most useful investigation to locate the site of bleeding.

Investigations :

1. Barium enema :

(a) In the prediverticular stage : A saw teeth appearance of the colon is present.
(b) Then fully developed diverticulae will be visualized.
(c) Barium enema can diagnose a concomitant lesion.

- **Barium enema is contraindicated during acute diverticulitis.**

2. Sigmoidoscopy : May detect the mouths of the diverticulae. Less accurate than barium enema & diagnose any concomitant lesion.

3. CT scan : best investigation in acute diverticulitis.

Treatment :

1. Stage of diverticulosis : A high fiber diet, antispasmodics.

2. Acute diverticulitis : Treated conservatively on the same principles as appendicular mass.

3. Acute diverticulitis is complicated by a pericolic abscess : Incision and drainage.

4. Acute diverticulitis complicated by generalized peritonitis : urgent laparotomy & resect the perforated colon by a Hartmann's procedure, then do peritoneal toilet and drainage.

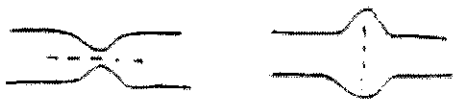
5. Chronic diverticulitis : colectomy is performed after adequate preparation.

6. Bleeding : resuscitation, usually bleeding stops spontaneously. If the bleeding is persistent, colectomy is performed (after adequate preparation).

Inflammatory bowel disease

The term **inflammatory bowel disease** is usually used to denote ulcerative colitis and Crohn's disease. However, bowel inflammation can be caused by other diseases e.g. colitis, amoebic colitis, ileocecal tuberculosis, and typhoid enteritis. Inflammatory bowel disease is not common in Egypt and some patients are wrongly diagnosed and treated as amoebic colitis.

Ulcerative colitis	Crohn's disease
Etiology	
Unknown, may be immunologic, genetic or environmental.	Unknown, heredity, focal ischemia may have a role.
Pathology	
Site : The disease begins at the dentate line of the anal canal and extends proximally.	**Site :** The commonest site is the distal ileum; the disease can affect any part from the mouth to the anal canal. Next common site is the colon, followed by the anal canal.
No skip areas of normal bowel.	It is characterized by skip lesions.
Only the mucosa and submucosa are involved with ulceration & pseudo polyps. **The extent of the disease varies from :** 1. Rectal involvement alone (proctitis). **to :** 2. Whole colon and rectum affection (pancolitis).	The inflammation involves the whole thickness from mucosa to serosa (transmural), as well as the mesentery which is also thickened and contains enlarged lymph nodes.
M.P.: Crypt abscesses.	**M.P.:** Granulomatous infiltration of the lymphatics of the submucosa with the presence of non-caseating giant cells.
Complications	
[A] Intestinal :	
1. Toxic megacolon may be fatal. **2. Hemorrhage.** **3. Colon cancer.** The risk is higher in patients with pancolitis for more than 10 years. Carcinoma on top of ulcerative colitis is often multicenteric. **4. Stricture.**	**1. Stricture.** **2. Abscess and fistula** to another bowel loop, bladder, vagina or to the skin. **3. Perianal abscess, anal fistula and fissure.** **4. Malabsorption** in extensive disease or with extensive intestinal resection.
[B] Extraintestinal :	
Arthritis, cholangitis, liver cirrhosis and skin lesions, finger clubbing.	
Clinical picture	
• Age : The third and fourth decades. • It is more common in females.	• Age : Second to fourth decades. • Males and females are equally affected.
• Watery diarrhea (mixed with blood, pus and mucus and accompanied by tenesmus), weight loss and dehydration occur to varying degrees. • The disease is characterized by the occurrence of remissions and relapses. • **Picture of complications.**	• Watery diarrhea (mixed with blood, pus and mucus and accompanied by tenesmus), weight loss and dehydration occur to varying degrees. • The disease is characterized by the occurrence of remissions and relapses. • There may be mass in the right iliac fossa. • **Picture of complications.**

Ulcerative colitis	Crohn's disease
Investigations	
1. Barium enema : Ulcers, as well as colonic shortening and loss of haustrations in long standing disease.	**1. Barium meal and follow through :** Segmental areas of stricture and cobble stone appearance of the mucosa caused by longitudinal fissures. It may also show internal or external fistulae. The finding of a narrowed terminal ileal segment is known as the **"string sign of Kantor".**
2. Colonoscopy and biopsy.	**2. Biopsy of accessible lesions** (e.g. colonic biopsy) proves the diagnosis.
Treatment	
[A] Conservative :	
1. Correction of anemia & hypoproteinemia. 2. Antispasmodics are given for pain. 3. Corticosteroids (either systemic or by enema) and antibiotics (Sulphasalazine or Flagyl are given in acute active disease).	1. Correction of anemia & hypoproteinemia. 2. Antispasmodics are given for pain. 3. Corticosteroids and antibiotics (Sulphasalazine or Flagyl are given in acute active disease).
[B] Surgical treatment is indicated for :	
1. Failure of medical management to control the disease. 2. Development of complications.	1. Failure of medical management to control the disease. 2. Development of complications.
The surgical options include : **1. Panproctocolectomy with permanent ileostomy.** **2. Subtotal colectomy and ileorectal anastomosis** & follow up for fear of development of a rectal carcinoma. **3. Subtotal colectomy with distal rectal mucosectomy.** An ileal pouch is fashioned to act as a reservoir, and is anastomosed to the anal canal within the left rectal muscles.	Method : • **Limited resection to the area that is causing the problem.** As the patient may be subjected to further resections in the future. • Localized strictures are treated by **stricturoplasty.**  *Fig. (38)*

Bilharzial colitis

Incidence :

More in young adult males living in endemic areas in Egypt.

Etiology :

Schistosoma mansoni, and less commonly by Schistosoma hematobium.

Pathology :

- The usual sites are the sigmoid colon and the rectum.
- Chronic inflammatory cells surround the deposited ova leading to the formation of Bilharzial granulomas.
- The disease occurs in two forms :

1. Submucous form :

- The mucous membrane becomes hyperemic, edematous and bleeds easily.
- In chronic cases the mucosa develops multiple polyps and later on ulcerations.
- The polyps may be diffusely arranged or they may be arranged in the form of a collar due to the circular arrangement of the veins.

2. Diffuse form :

- All layers of the colon as well as the mesocolon are affected.
- In addition to the mentioned mucosal changes, the sigmoid colon forms what is known as a **"Bilharzial pericolic mass".**

Complications :

1. **The condition is not precancerous** and does not produce strictures.
2. **Anemia** as a result of chronic blood loss.
3. **Hypoproteinemia** as a result of chronic protein (mucous) loss.
4. **Partial rectal prolapse.**
5. **Prolapse and strangulation of a polyp.**
6. **Complications of associated liver fibrosis and portal hypertension.**

Clinical features :

1. History of : Diarrhea, tenesmus, and the passage of mucus and blood in stools.
2. General examination : Clubbing of fingers may be present.
3. Local examination : The sigmoid colon is thickened and tender.

- In the diffuse form : a hard nodular mass is felt in the left iliac fossa. The mass may be mobile or fixed, and is often so hard as to simulate cancer (but features of intestinal obstruction are absent).

4. Rectal examination : Detects multiple polyps.
5. Picture of complications.

Investigation :

[A] Laboratory :

1. Stool analysis : shows blood, pus, and probably viable Bilharzial ova.
2. Urine analysis : may reveal associated urinary infestation.
3. Blood picture : may show anemia.
4. Liver function tests may reveal hypoalbuminemia.

[B] Sigmoidoscopy : shows the mucosal changes. Examination of fresh biopsy specimens, of ulcer scrapings shows the ova.
[C] Barium enema : may show multiple rounded filling defects of the polyps.

Treatment :

- All cases are initially treated by repeated courses of antibilharzial drugs. Oxamniquine or praziquantel.
- Polyps are treated by colonoscopic polypectomy at multiple sessions.
- Excision of an affected sigmoid colon is rarely needed.

Tuberculosis of the intestine

There are two forms of intestinal tuberculosis :

Ulcerative tuberculous enteritis	Hyperplastic ileocecal enteritis
Etiology	
• Secondary to pulmonary tuberculosis, & arises as a result of swallowing tubercle bacilli.	• Primary infection due to ingestion of tubercle bacilli (bovine or human type) by patients with high resistance.
Pathology	
• Multiple ulcers in the terminal ileum. • The long axis of the tuberculous ulcer lies transversely.	• The disease occurs most commonly in the ileocecal region. • There is much thickening of the intestinal wall, and narrowing of the lumen. • There is early involvement of the regional lymph nodes, which may caseate.
Complications	
• Stricture. • Perforation is rare.	• Stricture. • Abscess and fistula formation are rare.
Clinical picture	
• Attacks of abdominal pain, intermittent diarrhea and weight loss.	• Attacks of abdominal pain, intermittent diarrhea and weight loss. • Sometimes mass in the right iliac fossa.
• Picture of complications.	• Picture of complications.
Investigations	
	A barium meal and follow through reveals narrowing of the lumen of the affected area and an elevated cecum that may be subhepatic.
Treatment	
• In early cases treatment is by antituberculous drugs. • In patients with intestinal stricture, surgical excision may be needed.	• Is by antituberculous chemotherapy. • Right hemicolectomy with removal of the diseased ileum is indicated in obstructing lesions and if malignancy cannot be excluded.

Surgical complications of typhoid & paratyphoid

1. Intestinal hemorrhage.
2. Perforation around the third week.
3. Acute non-calcular cholecystitis.
4. Chronic cholecystitis (Carrier state).
5. Deep venous thrombosis.
6. Typhoid arthritis and ostemoyelitis.

Intestinal ischemia

Chronic intestinal ischemia

- **Atherosclerosis** is the main cause.
- There is postprandial abdominal pain, which starts 15-30 minutes after a meal, and lasts for about an hour. This pain is called **"abdominal angina".**
- **Aortography** is diagnostic.
- **Surgically treated** either by endarterectomy or by inserting a bypass dacron graft from the aorta to the superior mesenteric artery beyond the narrowing.

Acute intestinal ischemia

Acute ischemia of the intestine is a very serious surgical emergency that is highly fatal.

(A) Acute mesenteric vascular occlusion

It is commonly the superior mesenteric artery or vein that is occluded.

Etiology :

1. Mesenteric arterial embolism. The source may be: see embolism.
2. Mesenteric arterial thrombosis.
3. Mesenteric venous thrombosis.

Pathology :

1. Ischemic damage :

- Mucosa Sloughs within three hours and bleeds in the lumen.
- Within six hours the whole thickness of the intestinal wall is affected, and exudes serosanguinous fluid in the peritoneum.
- The intestine becomes gangrenous and soon perforates producing peritonitis.

2. Reperfusion damage :

- After the return of blood flow, either spontaneously or by surgery.

Clinical picture :

- Severe acute abdominal pain, vomiting, and bleeding per rectum.

Examination :

- In the early stage : there are little physical signs.
- Later : Picture of hypovolemic shock from blood loss and peritonitis form perforation.

Differential diagnosis: From other causes of acute abdomen mainly from acute pancreatitis.

Acute pancreatitis: The common features between acute mesenteric vascular occlusion and acute pancreatitis are severe pain, vomiting, minimal local signs in the early phase, and the occurrence of shock.

- **The distinction is important as the management of acute pancreatitis is essentially conservative, while that of acute mesenteric vascular occlusion is urgent surgery.**

Investigations :

1. Blood picture : There is marked leucocytosis.

2. Serum amylase is elevated.

3. Plain X-ray of the abdomen may show air-fluid levels in the proximal intestine, intramural gas or gas in the portal venous system.

4. The value of arteriography is controversial, and its use may delay surgical intervention.

Treatment :

Preoperative preparation :

1. Restoration of blood volume and parenteral antibiotics.
2. Nasogastric decompression by a Ryle's tube.

Laparotomy :

- **Resect the gangrenous intestine** and restore blood flow of viable ones by embolectomy for embolism and by endarterectomy or bypass for thrombosis.
- **Primary anastomosis is better avoided in** cases of peritonitis, doubtful viability and in patients with bad general condition.
- For patients who receive a primary anastomosis **a second-look operation after 24 hours** is advised by some surgeons to check the viability of the intestine.

Prognosis :

- Mortality rate is 30 - 45%.
- Up to 70% of the small intestine can be resected without major nutritional consequences.
- Patients who have resection of longer segments develop the **"short bowel syndrome"** its main feature is **malabsorption.**

(B) Non-occlusive intestinal ischemia

- It occurs with low cardiac output states as arrhythmias and major sepsis.
- Splanchnic vasoconstriction occurs inducing intestinal ischemia that may lead to gangrene.
- There is no block to the mesenteric vessels.
- **The mortality is 90% as the condition affects critically ill patients.**

(C) Focal intestinal ischemia

Etiology :

1. Vascular problems as embolism, arterial thrombosis, and trauma.
2. Strangulated hernia, adhesions, and volvulus. Ischemic colitis is one type that deserves a special attention.

Ischemic colitis

- It is a type of focal ischemia.
- Patients are usually 50-70 years old
- **Etiology :**

1. A drop of blood pressure.
2. Surgery on the abdominal aorta if the inferior mesenteric artery is ligated.

- **Site : The splenic flexure is the most commonly affected.**
- **Clinical picture :** Acute abdominal pain and the passage of blood per rectum.
- **Fate :**

1. Complete resolution.
2. Gangrene, perforation and peritonitis.
3. Stricture.

Intestinal Obstruction

Definition :

Arrest of downward propulsion of intestinal contents.

Classifications :

There are different types of classification of intestinal obstruction.

[A] According to the pathological nature of cause :

1. Simple mechanical obstruction.
2. Strangulation obstruction : Characterized by significant impairment of the blood supply of the involved bowel segment. If not relieved within 6 hours, will lead to gangrene.
3. Paralytic ileus : is due to loss of propulsive power of the bowel leading to functional obstruction.

[B] According to the level of obstruction :

1. High small bowel obstruction.
2. Low small bowel obstruction.
3. Large bowel obstruction.

[C] According to the onset and course of obstruction :

1. Acute obstruction.
2. Chronic obstruction e.g. colon cancer, the symptoms are insidious and slowly progressive. The patient has constipation and distention.
3. Acute on tope of chronic obstruction.

NB. **Pseudo-obstruction** includes a group of disorders which give the clinical picture of **I.O**

Common causes of intestinal obstruction according to age	
Neonates	Congenital atresia, volvulus neonatorum, anorectal malformations, mechonium ileus, and Hirschsprung's disease.
Infants	Ileocecal intussusception, Hirschsprung's disease, and strangulated hernia.
Adults	Adhesions, strangulated hernia, rectal carcinoma.
Elderly	Colon carcinoma, strangulated hernia, volvulus sigmoid.
Strangulated hernia, adhesion and paralytic ileus are common causes in different groups.	

Acute mechanical intestinal obstruction
General principles

Etiology : The cause of obstruction may be :

1. In the lumen : e.g. fecal impaction, gall stone ileus and parasitic infestation.

2. In the wall : e.g. congenital atresia, tumors, Crohn's disease, chronic diverticulitis.

3. Outside the wall : e.g. adhesions (commonly post-operative), strangulated hernia, and volvulus.

Pathology :

[A] Simple obstruction :

Distal to the obstruction : the intestine empties and becomes collapsed.

Proximal to the obstruction :

1. The intestine becomes distended by gas and fluid :

(A) Gaseous distention is due to :	(B) The origin of fluid :
1. Swallowed air (68%). 2. Diffusion from congested vessels (22%). 3. Bacterial fermentation (10%).	1. GIT secretions (8000 cc/24 hours) : Saliva (1500cc), gastric secretions (2500 cc), bile and pancreatic juice (1000 cc) and intestinal juice (3000 cc). 2. Intake of water and fluids. 3. Transudation of fluids from blood vessels.

2. There is hyperperistalesis in an attempt to overcome the obstruction.

3. Distension impairs blood supply, and may end in ulceration and perforation :

(a) In cases of colon obstruction with a competent ileocecal valve, the rising pressure in the closed proximal colon segment (closed loop obstruction) causes perforation of the cecum.

(b) Perforation may also occur from the pressure of an adhesion hand or the edge of a hernia defect on the bowel wall.

[B] Strangulation obstruction : In addition to the above :

- Mucosa Sloughs within three hours and bleeds in the lumen.
- Within six hours the whole thickness of the intestinal wall is affected, and exudes serosanguinous fluid in the peritoneum.
- The intestine becomes gangrenous and soon perforates producing peritonitis.

[C] General lethal effects (with both A & B) :

1. Fluid and electrolyte loss from vomiting and from accumulation in the proximal bowel.
2. Septicemia from peritonitis.

Clinical features :

I- Symptoms :

The following are the cardinal symptoms of acute mechanical intestinal obstruction, but are not necessarily present in all patients.

1. Pain : Usually the first symptom. It is colicky and is caused by the hyperperistalesis.

2. Distention.

3. Absolute constipation : (obistipation) Failure to pass flatus in addition to stools.

4. Vomiting : In neglected cases it becomes greenish then brown, offensive & feculent (not fecal).

Items	High small intestine	Low small intestine	Large intestine
Pain	Above umbilicus	Around umbilicus	Peripheral & lower abdomen
Vomiting	Very early	After few hours	Late
Distension	Minimal or absent	Moderate & central	Huge & peripheral
Absolute constipation	Late	After few hours	Early
Shock	Severe	Moderate	Minimal

II- Clinical examination :

1. General examination : Evidence of dehydration as tachycardia, oliguria, dry tongue, or even hypotension may be present.

2. Abdominal inspection :

- Distension and visible peristalsis.
- Strangulated external hernia should be looked for.
- Scars of previous abdominal surgery may denote intra-abdominal adhesions.

3. Abdominal palpation : A mass may be felt (tumor or intussusception).

4. Auscultation : Accentuated intestinal sounds.

5. Rectal examination : Reveals an empty rectum. An exception is the finding of a hard fecal mass in an elderly bed-ridden patient causing fecal impaction.

III- Strangulation is suspected with :

1. Toxic patient, tachycardia, fever, and leucocytosis.
2. Evidence of blood loss as pallor, tachycardia, and hypotension.
3. Pain which is not relieved by naso-gastric suction. (Decompressing the bowel doesn't relieve ischemic pain).
4. Marked tenderness, rebound tenderness and rigidity.

Items	Simple occlusion	Strangulation	Paralytic ileus
Pain	Colicky, intermittent	Sever, persistent	No pain
Shock	Minimal & later	Early & sever	Moderate
Palpation	-ve	Tenderness, rebound tender & rigidity	-ve
Auscultation	Loud exaggerated intestinal sound	First exaggerated sound then disappears	Dead silent abdomen
Nasogastric suction	Relieve pain	No effect on pain	Relieve distention

IV- Picture of complications :

1. Shock : Late except in strangulation, may be neurogenic from pain, hypovolemic, or septic.
2. Fluid and electrolytes imbalance : due to vomiting and defective absorption → hypovolemic shock and hypokalemic hyponatremic acidosis.
3. Toxic absorption from the retained fluid and from the necrotic tissue. This may occur after operative release of the obstruction (as the obstruction is present they are not absorbed). This is avoided by continuous Ryle tube suction.
4. Strangulation : due to secondary interference of the blood supply.
5. Perforation of the distended loops → peritonitis.
6. Respiratory complications.

Investigations :

- Are needed to confirm the diagnosis of intestinal obstruction, to define its level and cause, and to estimate the severity of water and electrolyte imbalance.

[A] Blood urea and electrolytes.

[B] Blood picture.

[C] Plain X-ray of the abdomen :
1. Erect film : Multiple gas-fluid levels.
2. Supine film : The level of obstruction is known by the gas pattern of the distended proximal intestine:

- Distended jejunal loops show the characteristic circular mucosal folds (valvulae conniventes) crossing from one side of the lumen to the other.
- Distended ileal loops appear as structureless tubes with no mucosal pattern.
- A colon full of gas shows haustrations that do not appear to reach the other side of the lumen.

[D] Ultrasound examination : can reveal distended bowel loops. A mass of intussusception can also be demonstrated.

[E] Double enema test : The first enema is given and its result is neglected and the second enema is given, after ½ hour. In absolute constipation the second enema brings fluid only without force, feces or flatus and may be blood stained in intussusception, volvulus or mesenteric vascular obstruction.

[F] Barium enema : To detect any colonic obstruction.
[G] CT scan with contrast: has a sensitivity of 80-90% for the diagnosis of bowel obstruction.
N.B.: Barium meal is contra-indicated as it worsens the condition.

Treatment :

The treatment is urgent relief of obstruction, usually by surgery, after adequate preoperative preparation.

[A] Pre-operative preparation "Drip and Suck" :

1. IV replacement of fluid & electrolytes together with blood & plasma if the patient is shocked.
2. Gastric aspiration by a naso-gastric tube to decompress the bowel and to reduce the risk of inhalation during induction of anesthesia.
3. Antibiotics : If there is a possibility of strangulation.
4. A Foley's catheter is inserted to check the urine output.

[B] Conservative treatment : may be successful in certain situations provided that the case is early and there is no clinical evidence of intestinal ischemia e.g.

1. **Adhesive intestinal obstruction.**
2. **Ileocecal intussusception.**
3. **Sigmoid volvulus.**
4. **Fecal impaction** is treated by enema to dissolve the obstructing hard fecal mass.

[C] Operation : The steps of the operation are :

- A longitudinal exploratory incision.
- Look at the cecum. If it is collapsed, this denotes small gut obstruction. If it is distended, this means large bowel obstruction.
- Determine the level of obstruction which is the junction of dilated and collapsed bowel loops.
- Relief of obstruction e.g. by division of adhesive bands, or untwisting of volvulus.
- Bowel viability is assessed.
- Non-viable intestine is resected.
- Small intestinal ends are anastomosed (primary anastomosis).
- Colon (see principles of colon surgery).

Special forms of acute mechanical intestinal obstruction

Neonatal intestinal obstruction

Major types of intestinal obstruction :

1. Duodenal obstruction : Atresia, stenosis, diaphragm, malrotation with bands or volvulus (volvulus neonatorum) and annular pancreas.

2. Jejunal and ileal obstruction : Volvulus neonatorum, atresia, stenosis, meconium ileus, strangulated inguinal hernia, intussusception, peritoneal bands or hernia and duplication cysts.

3. Large bowel obstruction : Hirschsprung's disease, anorectal anomalies & meconium plug syndrome.

4. Necrotizing enterocolitis.

5. Adhesive obstruction following surgery : e.g. post-appendicectomy.

6. Paralytic ileus : e.g. due to hypokalemia or chest infection.

Duodenal atresia : Duodenal obstruction usually occurs at the level of ampulla of Vater Causing bilious vomiting. 30% of cases are associated with Down's syndrome & 20% with congenital heart disease.

- Duodeno-duodenostomy is the operation of choice.

Volvulus neonatorum (midgut volvulus) : The baby is born with a free midgut having a narrow-based mesentery, and is, therefore, liable to twist around its mesenteric axis. The result is strangulation obstruction of whole midgut. There is usually a fibrous band (Ladd's band) which can obstruct duodenum.

- Urgent surgery is needed to untwist the gut to divide the band.

Jejunal & ileal atresia : Due to a vascular insult to the intestine in-utero.

- Treatment : Resection of the atretic segment is done with bowel anastomosis.

Meconium ileus : There is obstruction of ileum due to thick inspissated meconium.

Mid Gut Rotation

- At the 4th week of intra-uterine life, the mid gut extrudes into the umbilical cord.
- Reduction of this loop occurs later on through three stages:
  a) **1st stage:** 90° anticlockwise rotation so, small intestine rests on the right.
  b) **2nd stage:** 180° anticlockwise rotation so, small intestine rests on left and caecum is below liver.
  c) **3rd stage:** descent and fixation of the caecum to the posterior abdominal wall.

Large bowel obstruction
Hirschsprung's disease (aganglionic megacolon)

It is one of the commonest causes of intestinal obstruction in infancy.

Etiology :

It is due to failure of cephalo-caudal migration of the parasympathetic plexus into the distal bowel, interfering with the normal peristaltic propulsive wave leading to obstruction, stasis and recurrent attacks of entero-colitis.

Pathology :

The aganglionic segment starts in the anus and affects the recto-sigmoid in 75% of cases but may extend proximally in a minority of cases to affect the whole colon.

Clinical picture :

- **Any newborn presenting with delayed passage of meconium for more than 24 hours should be considered as having Hirschsprung's disease until proved otherwise.**

- The infant is suffering from chronic constipation. Defecation occurs only after insertion of a suppository or doing an enema.
- Progressive abdominal distention.
- The growth and development of the patient will be affected.
- Per-rectal examination reveals an empty rectum and when the finger is removed, it is followed by a sudden gush of gases or stools.

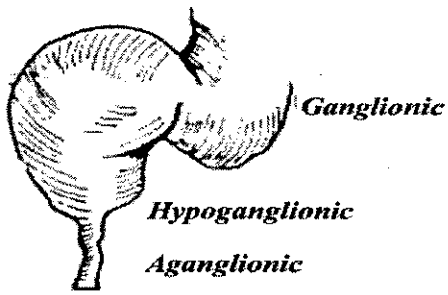
- **It may present with picture of complication :**

1. **Acute obstruction.**
2. **Obstructive enterocolitis :** The patient develops pyrexia, abdominal distention and diarrhea.

Investigations :

1. Barium enema :

- Needs to be done without preparation and a small amount of barium administered.
- It shows the stenosed aganglionic segment, followed by a funnel due to a segment of incomplete ganglia, followed by a dilated normal ganglionic bowel.



2. Rectal biopsy :

- Only a small segment of the mucosa and submucosa is taken by a special forceps.
- Ganglion cells in the submucous layer are absent.

Treatment :

The aim of treatment is to do complete excision of the aganglionic segment followed by a colo-anal anastomosis. This is a major operation which needs good general condition & a prepared colon.

Ano-rectal malformations

Classifications :

1. High anomalies : The rectum ends above the level of the pelvic floor and usually has a fistulous communication with the viscus lying anteriorly i.e. the posterior urethra in males & the vagina in females.
2. Low anomalies : The rectum has passed through the pelvic floor and opens onto the surface in an abnormal position which is usually anterior to the normal anus.

- **Congenital anomalies are often found in association with ano-rectal malformations** such as vertebral anomalies, urinary tract malformations, gastro-intestinal and cardiovascular defects. They are more common in the high than in the low types.

Clinical features :

- Picture of neonatal Intestinal obstruction.
- It is easy to be diagnosed provided the anus has been inspected and examined.
- Differentiation between the different types of ano-rectal malformation is more difficult and is dependent on experience and further investigation.
- Meconium on the tip of the penis are indicative of high defects with recto-urethral fistula.
- **In the male :** in the covered anus, white or green meconium may be seen anterior to the anus.
- **In the female :** a careful search must be made for any ectopic opening in the vulvar, vestibular or perineal regions.

Investigation :

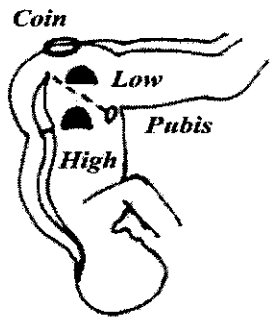
Invertogram :

- Lateral radiograph of the infant upside down, centered on the greater trochanter and with a radio opaque marker on the anus.
- A line is then drawn between the symphysis pubis and the tip of the sacrum.
- If a shadow of gas in the rectum is seen :

1. Above the pubo-coccygeal line (P-C line) : the anomaly is low one,
2. Below the pubo-coccygeal line (P-C line) : the anomaly is a high one.

- **Also Comment on :**

1. Air in the bladder.
2. Sacral anomalies.



Invertogram

Operations :

1. High anomalies : Treated with a temporary colostomy, a second stage abdomino-ano-rectal pull through, to be followed by a third stage closure of colostomy.
- In selected patients a primary single stage abdomino-ano-rectal pull-through operation is satisfactory.

2. Low anomalies : a single stage perineal operation gives excellent results.

N.B: early detection of neonatal intestinal obstruction

1) ***Antenatal:*** polyhydraminos;raises the suspicion of high gut obstruction.
2) A neonate with bilious gastric aspirate more than 25 ml
3) Any baby presenting with persistent, bile stained vomiting should be considered to have intestinal obstruction until proved otherwise.

Intussusception

Definition :

- Invagination of segment of the bowel (intussusceptum) into the lumen of the adjacent one (intussuscepient).

- It is one type of strangulation obstruction because the mesenteric vessels of the inner layer are stretched and compressed by the outer layer.

Anatomical types :

1. Ileocecal intussusception : The commonest, ileum is invaginated into the cecum with the ileocecal valve forming the apex.
2. Ileo-ileal : Ileum is invaginated into the ileum.
3. Ileo-colic : Ileo-ileal intussusception advancing till its apex enters the colon.
4. Colo-colic : Colon is invaginated into the colon.
5. Retrograde : As jejunogastric intussusception after gastrojejunostomy.

Clinical types :

1. Infantile types : The commonest, usually ileocecal type.
2. Adult type : Rare, due to organic lesion at the apex as polypoid tumor or parasites.

Etiology :

- The infantile ileocecal intussusception is idiopathic i.e. the cause is not known.

- It is possible that an adenovirus causes swelling of the lymphoid follicles in the terminal ileum, and these protrude into the lumen acting as a foreign body which is forced distally along the gut. This may explain the frequent occurrence of intussusception at the age of weaning.

- In a minority of cases there is an evident cause at the head of the intussusceptum e.g. polyp, Meckel's diverticulum, or submucous hematoma in a patient with Henoch Schnolein purpura. This occurs mainly in adults. In elderly persons carcinoma of the colon may cause colo-colic intussusception.

Pathology :

- **The ileocecal valve is invaginated into the cecum** and may travel along the ascending, transverse, descending colon and may protrude through the rectum and anus.

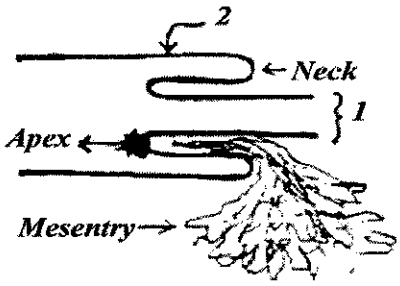
- **An intussusception consists of :**

1. Intussusceptum : Consists of an entering layer and a returning layer.

2. Intussuscepient : Is the outer ensheathing layer.

3. Apex : Is the junction between the entering and the returning layer. It is always constant at the ileocecal valve.

4. Neck : Is the junction between the ensheathing and returning layer.



- **The mesentery containing the blood vessels** may be compressed between the entering and the returning layer → ischemia and gangrene.

Clinical picture of infantile ileocecal intussusception :

1. The common age is between 3 and 12 months. **Male : Female** = 2:1.
2. Pain : Attacks of colicky pain denoted by screaming, drawing the legs up to the abdomen, and pallor.
- The attacks alternate with intervals of apparent wellbeing.

3. Vomiting.
4. Passage of bloody mucus (that looks like red current jelly) per rectum.
5. Distention is not an early feature.
6. There is emptiness of the right iliac fossa (Sign de Dance).
7. A sausage shaped mass felt anywhere except in the right iliac fossa is diagnostic. It may not be felt if it is hidden under the costal margin.
8. Rectal examination reveals bloody mucus & sometimes the head of the intussusception can be felt.
9. Picture of strangulation.
10. Neglected cases show evidence of peritonitis with marked distention.

Any infant having colicky abdominal pains with the passage of bloody mucous per rectum, should be suspected of having intussusception.

D.D.:

1. Gastroenteritis : There is fever, vomiting, diarrhea; shock is very late, no mass felt on abdominal or P-R examination.
2. Rectal prolapse : No manifestations of intestinal obstruction, a finger cannot pass around the protruding mass.
3. Henoch-Schnolein purpura.

Investigations :

1. Blood picture may show evidence of anemia & electrolyte disturbance.
2. In cases of doubtful diagnosis a barium enema is performed. The barium will fill the colon until it stops at the area of intussusception where a cylindrical filling defect appears and there is arrest of further progress of the contrast **(Claw sign or Meniscus sign, and Coiled spring sign).**

Treatment of infantile ileocecal intussusception :

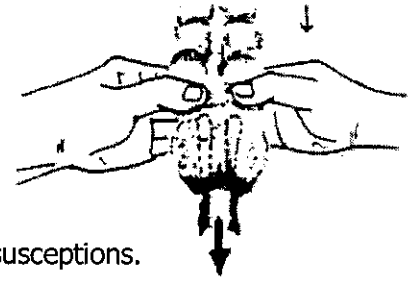
Hydrostatic reduction :

Indications : Early cases that show no evidence of peritonitis.
Methods : Barium (or air) is run in per rectum and followed radiologically under the screen.
- The diagnosis is established and the reduction, affected by the pressure of the barium column, is monitored until it is complete. This is confirmed by visualization of appendix and the terminal ileum.

Contraindication : Late cases or in the presence of abdominal distention or rigidity.
Complications : Reduction of a gangrenous intussusception or perforation of the bowel.

Surgery :

Indications : When hydrostatic reduction fails or the condition is advanced from the start.
Method :



- At laparotomy, the head of the intussusception is squeezed backwards out of the containing colon.
- The proximal ileum should never be pulled backwards as this may lead to intestinal tears.
- The presence of gangrene or irreducible intussusception is an indication for bowel resection and anastomosis.
- Important to check the whole bowel as there may be multiple intussusceptions.
- **Mention differences between viable & non viable gut.**

Prognosis :

Mortality is high in gangrenous cases. Intussusception may recur in minority of cases.

Adhesive intestinal obstruction

Intraperitoneal adhesions constitute the commonest causes of intestinal obstruction in developed countries.

Etiology :

- They result from previous abdominal surgery or may be Post-inflammatory adhesions e.g. after septic or tuberculous peritonitis.
- Their exact etiology is obscure.
- It is commoner when talc is used to lubricate the surgical gloves.
- Adhesions develop at the sites of ischemia of the bowel or at the scars of the abdominal wall.

Pathology :

- Adhesions may be multiple or solitary.
- They induce obstruction by kinking or by directly obstructing a small intestinal loop.
- Strangulation may result from compression of the loop's blood supply. A band may include localized ischemic necrosis by direct pressure on the intestine at the level of block.
- Adhesions have a tendency for recurrence producing recurrent intestinal obstruction.

Clinical features :

- Those of acute or recurrent acute small bowel obstruction.
- Almost always there is a scar of previous abdominal surgery.

Treatment :

[A] Conservative treatment :

Indications : Early cases with no evidence of strangulation.

Method :

- Naso-gastric suction, and intravenous replacement of losses (drip and suck).
- Close observation is necessary to judge success which is indicated by resolution of pain and distention, passage of flatus, and the retrieval of clear gastric aspirate.

[B] Surgery :

Indication :

1. Conservative treatment fails.
2. If there is evidence of strangulation or gangrene.

Method :

- Adhesions are divided and the bowel viability is assessed and dealt with accordingly.

Volvulus

Definition : Twisting of loop of bowel around its mesenteric axis.

Pathology :

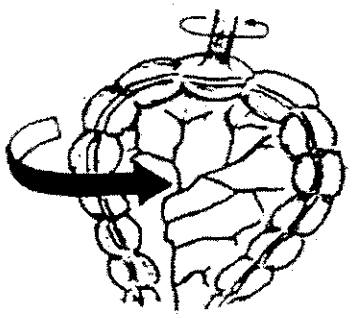
- It results in a combination of obstruction together with occlusion of the main vessels at the base of the involved mesentery.
- It is, therefore, a form of strangulation obstruction and of closed loop obstruction.
- A closed loop is formed within which the pressure rises rapidly, with increasing risk of gangrene and perforation.

• **Site :** Commonest in the sigmoid colon. It may also affect the cecum, stomach, the small intestine and Midgut volvulus of the neonates.

• The upper loop usually falls in front of the lower so that the twist is almost always in anticlockwise direction → **closed loop obstruction leading to :**

1. Huge distension of the sigmoid colon with gas and fluid (diffusion from congested bowel).
2. The colon above is distended.
3. Rectum below is collapsed and empty.

• Interference with the blood supply ultimately occur if it rotates more than 1.5 turn → strangulation and perforation follows with rapid fatal peritonitis.



Predisposing factors to sigmoid volvulus :

1. A long sigmoid colon.
2. A narrow base of sigmoid mesocolon
3. A heavily loaded sigmoid as a result of chronic constipation.
4. Adhesions at the apex of the sigmoid loop facilitate its twisting.

Clinical picture :

1. Sigmoid volvulus is commoner in elderly chronically constipated males.

2. The features are those of acute colon obstruction (discuss) with :

• Marked distention because of the accumulation of fluid and gas in the closed sigmoid loop.
• Pain in the hypogastrium.
• Vomiting : Late.
• Absolute constipation : early.

3. Picture of strangulation.

4. P/R examination : Empty rectum, blood may be found on tip of examining finger.

5. Neglected cases show picture of complications : Peritonitis, shock (hypovolemic or septic) and electrolyte disturbance.

Investigations : As general.

• **Plain X-ray** shows the huge gas-filled sigmoid loop that may look like the inner tube of car tire **(Omega loop).**

Treatment :

[A] Conservative treatment :

Indication :

• Early cases with no evidence of gangrene.

Method :

• Rectal tube is passed through a sigmoidoscope to untwist the sigmoid loop.
• Success will lead to gush of gas and fluid stools.
• The tube is left in place and the patient prepared for later elective resection of the long sigmoid to prevent recurrence.

[B] Emergency Surgery :

Indications :

1. Failure of conservative.
2. Late presentation.

Method :

• Gangrenous bowel is resected, the proximal colon end is brought out to the skin as a terminal colostomy and the distal end is closed by sutures (Hartmann's procedure); for later elective anastomosis.
• Viable sigmoid is untwisted and either fixed to the posterior abdominal wall or resected as for gangrenous cases.

Paralytic ileus

- The Greek word ileus = to roll. It has been applied to colic.
- When one means paralytic ileus, it is inappropriate to use the short term ileus, as this condition lacks the colicky pain characteristic of mechanical obstruction.

Etiology :

1. Reflex inhibition of intestinal motility following abdominal operations, spine fractures, and retroperitoneal hemorrhage. This may be due to sympathetic over action.
2. Metabolic abnormalities as hypokalemia, uremia, and diabetic ketoacidosis.
3. Peritonitis due to direct toxic effect on the nerve plexuses of the intestine.
4. Drugs as anticholenergics (e.g. probanthine).

Pathology :

- There is marked distension of the small and large intestines with gas and fluid.
- The patient suffers a severe loss of fluid & electrolytes in the distended bowel & through vomiting.

Clinical features : As general

- The patient, does not have colicky abdominal pains, there is only **a sense of fullness** and discomfort.
- **Silent abdomen.**

Investigations :

- **Plain X-ray of the abdomen :** Multiple gas fluid levels. The gaseous distention includes the whole small and large intestines.
- **Blood urea and electrolytes.**

Prevention :

1. Preoperative correction of biochemical disturbances.
2. Gentle handling of the intestine during surgery.
3. For major abdominal surgery, a naso-gastric tube is used to decompress the bowel postoperatively.

Treatment :

1. Drip and Suck are the essentials of treatment.
2. Correction of underlying metabolic abnormalities and hypoproteinemia.
3. If a postoperative ileus is unduly prolonged for more than 3 days, one should think of peritonitis from a leaking intestinal anastomosis and of mechanical obstruction from early fibrinous adhesions. Both Conditions necessitate reoperation.

N.B: pseudo-obstruction:

a) This term includes a group of disorders which give the clinical picture of intestinal obstruction without organic lesions.
b) May be caused by many disorders e.g.elderly patients with cerebro vascular accidents,myxedema,drugs,uremia, or retroperitoneal irritation

Colorectal tumors

Tumors of the colon and the rectum share common properties.

Polyps of the colon and rectum :

	Type	Solitary	Multiple
Epithelial	Neoplastic	Adenoma	Familial polyposis coli (FPC), Gardner's syndrome
		Carcinoma	
	Hamartomas	Juvenile polyp	Multiple juvenile polyps
			Peutz Jegher's polyps.
	Inflammatory		Ulcerative colitis (pseudopolyps) Bilharzial polyps
	Unclassified	Hyperplastic polyp	Multiple hyperplastic polyps
Mesenchymal	Lipoma, leiomyoma, or hemangioma.		

Malignant tumors :

[A] Primary :

1. Carcinoma.
2. Carcinoid tumor.
3. Sarcomas.

[B] Secondary : These are the result of invasion from a nearby malignant tumor.

Familial Polyposis Coli (FPC)

Etiology :

- It is an autosomal dominant disease.
- 50% of the children of affected parents have this disorder.

Pathology :

- The colon and rectum are full of multiple polyps (at least 100).
- If untreated, carcinoma develops in 100% of cases by the fifth decade.

Gardner's syndrome is a variant of FPC. In addition to the above, there are polyps in the rest of the gastrointestinal tract, osteomas, cyst, soft tissue tumors & desmoid tumors of the abdominal wall.

Clinical features :

- Polyps usually present between the ages of 10 and 15 years.
- The commonest symptoms are diarrhea, bleeding, and abdominal pain.

Investigation :

1. Barium enema shows multiple rounded filling defects.
2. Sigmoidoscopy and biopsy.

Treatment :

1. Treatment of the effected person : Surgical options are similar to those for ulcerative colitis. (Pan proctocolectomy with terminal ileostomy).

2. The family members : Should be periodically examined by colonoscopy.

Carcinoma of the colon & Rectum

Incidence :

- **Age :** The general peak incidence of carcinoma of the colon & rectum is in the seventh decade of life; however rectal carcinoma is not uncommon in young adults.
- **Sex :** Cancer of the rectum & sigmoid colon is commoner in males, while cancer of the cecum is commoner in females.

Etiology :

1. Benign adenoma : particularlly villous adenoma.
2. Familial polyposis coli and Gardner's syndrome : The risk is 100% in untreated cases.
3. Ulcerative colitis : The risk is highest with pancolitis of more than 10 years duration.
4. The disease is more common in Western countries probably due to lack of high fiber diet.
5. After uretro-colic anastomosis.

Pathology :

Site :

- Two thirds in the rectum and the sigmoid colon.
- 25% Occur in the cecum & Right colon.
- Multiple tumors occur in 5% of cases.

Gross picture :

1. Cauliflower-like polypoid variety arises more commonly in the cecum.
2. Ulcerative type.
3. Stricture schirrous type arises more commonly in left colon.

Histologically :

- The tumor is an adenocarcinoma that arises from the columnar epithelium. It may be well, moderately, or poorly differentiated.
- Colloid carcinoma occurs in younger patients, and has a poor prognosis.

Spread :

1. Direct spread occurs first in the wall and then to the neighboring organs e.g. the urinary bladder.

- **The strong fascia of Denonvillier lying in front of the rectum retards the spread of rectal cancer to the bladder.**

2. Lymphatic spread :

- In right & transverse colon :

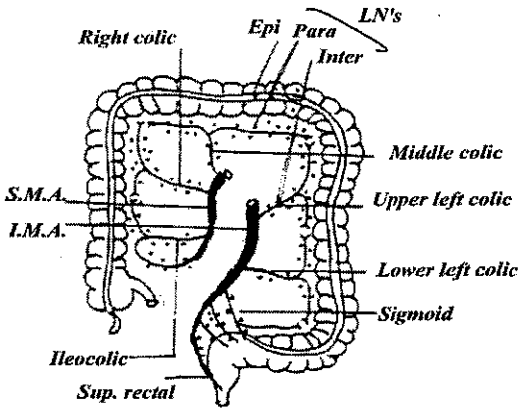
Epicolic, paracolic and then to superior mesenteric lymph nodes.

- In left colon :

Epicolic, paracolic and then to inferior mesenteric lymph nodes.

3. Blood stream spread :
Produces liver metastases in 20% of patients coming to surgery.

4. Transperitoneal spread :
Leads to peritoneal nodules and ascites.



Staging : Duke's staging

A	The growth is limited to the bowel wall.
B	The growth extends outside the bowel wall, but no metastasis to lymph nodes.
C	There are secondary deposits in the regional lymph nodes.
C_1	The local para-rectal, or para-colic lymph nodes alone are involved.
C_2	The superior or inferior mesenteric nodes.
D	Distant metastasis (not in the original Duke's).

TNM staging of colorectal cancer

Tumor	T1	invades into the mucosa
	T2	invades into the submucosa
	T3	invades to the subserosa but not breaching the serosa
	T4	invades serosa or another organ
Nodes	N0	no nodes involved.
	N1	1-2 nodes involved.
	N2	3 or more nodes involved.
Metastases	M0	no metastases
	M1	metastases.

Complications :

1. Intestinal obstruction : especially with left colon tumors due to :
(a) The smaller lumen of the left colon.
(b) Stool tends to be more solid.
(c) Carcinoma tends to be of the stenosing variety.
2. Perforation, enterocolic or vesicocolic fistula.
3. Bleeding : Although chronic bleeding is the rule, massive bleeding is rare.
4. Complications due to spread : e.g. jaundice, liver failure, and ascites.

Clinical features :

[A] Right colon cancer :

1. The patient may present with : anemia, weakness & loss of weight (**A**nemia, **A**norexia & **A**sthenia).
2. Pain : Recurrent attacks of pain in the right iliac fossa.
3. Hard mass in the right iliac fossa : It is differentiated from appendicular mass by the long duration and absence of toxemia and tenderness.
4. Obstruction : The patient does not present by intestinal obstruction as the lesion is usually of the cauliflower variety, the contents are liquid and the lumen of the colon is wide. Obstruction occurs, rarely, if the lesion obstructs the ileocecal valve.
5. Metastases.

[B] Left colon cancer :

1. The patient may present by change of bowel habits : Usually as progressive constipation, but there may be diarrhea or attacks of constipation alternating with diarrhea.
- **Spurious diarrhea** (early morning slime) may be present.

2. Large bowel obstruction : The patient may present as acute, subacute or chronic large bowel obstruction. There is constipation, severe abdominal distention but vomiting is late.
3. Bleeding per rectum : See before.
4. Mass in the left side of abdomen : As the lesion is usually of the infiltrating schirrous variety, it rarely presents by a mass. If a mass is palpable, it is usually due to impacted stools above the obstruction.

[C] Rectal cancer :

Often presents with picture of left colon with the following differences :
- **Bleeding per rectum.**
- **There may be tenesmus** (sense of painful incomplete evacuation), and the passage of mucus.
- **P/R examination** allows palpation of lesions that lie within 10cm of the anal verge.

Whenever rectal bleeding occurs in middle aged or older individual, even in the presence of hemorrhoids, coexisting rectal cancer must be ruled out.

General examination (for all sites) :

1. Check : the nutritional status, presence of anemia, and jaundice.
2. Abdominal examination : for ascites, or hepatomegaly.
3. Rectal examination : for presence of pelvic deposits.

D.D.:

1. Carcinoma of cecum : Causes of mass in the right iliac fossa.
2. Carcinoma of the transverse colon : Causes of mass in the epigastrium (see carcinoma of the stomach).
3. Carcinoma of the left colon & rectal cancer :

(a) Causes of mass in the left iliac fossa :
- Bilharzial mass (diffuse bilharzial colitis).
- Pericolitis in diverticulosis coli.
- Mass of stools in chronic constipation.
- Spastic colon.

(b) Other causes of bleeding P/R.
(c) Other causes of intestinal obstruction.

Investigations :

1. Blood picture : shows microcoytic hypochromic anemia.

2. Sigmoidoscopy & colonoscopy :
- Essential in all patients with altered bowel habit or rectal bleeding, and is mandatory in patients above the age of 40 who complain of piles.
- Biopsy is obtained from suspicious lesions.

3. Barium enema :
- Fixed irregular filling defect.
- Annular strictures of the left colon show a characteristic **"apple core appearance".**
- In rectal cancer, it is useful to exclude a second higher tumor.

4. CT scan with contrast: has a sensitivity of 80-90% for diagnosis of bowel obstruction.
5. To detect spread : liver function tests, abdominal ultrasound ,chest X-ray and IVU.
6. Carcinoembryonic antigen (CEA) :
- Is a tumor marker whose serum level is high in colorectal cancer but is not specific.
- It is of prognostic rather than diagnostic value.
- The level drops after a successful radical surgery.
- If it shows a rise in the follow up period, this signifies recurrence.

6. For patients presenting with acute intestinal obstruction :
- Plain X-ray of the abdomen, blood picture, blood urea, and electrolytes are needed.
- A barium enema in these circumstances can assure the diagnosis.

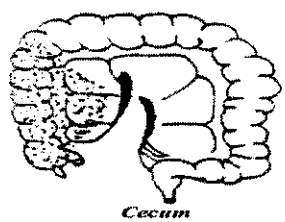
Treatment :

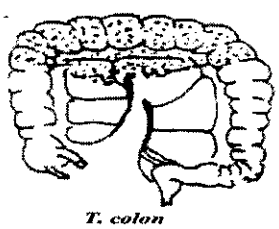
Surgery is the main line of treatment. Radical resection is the only curative measure.

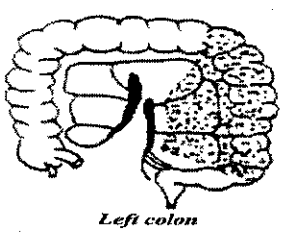
[A] Unobstructed operable (potentially curable) cases :

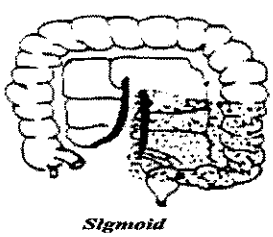
- Should receive **elective radical resection** (Removing the tumor bearing segment together with its lymphatic drainage area, in one mass and then to restore bowel continuity).

- Since the lymph nodes are so close to the main blood vessels arising form the superior and inferior mesenteric, their clearance requires ligation and division of these vessels at their origin, and consequently the whole part of the colon supplied by the removed vessels should be resected.


Cecum


T. colon


Left colon


Sigmoid

Site	Procedure	Vessels ligated	Structure removed
Tumors of cecum	Right hemicolectomy	The ileocolic, and right colic vessels are divided at their origin	The terminal 10 inches of the ileum, the cecum, the ascending colon, the hepatic flexure and the right half of the transverse colon are resected. The peritoneum of the posterior abdominal wall between the colon and the superior mesenteric that contains the divided arteries and veins, and the related lymph vessels and nodes are removed in continuity. The operation is completed by performing an ileotransverse anastomosis.
Tumors of transverse colon	Transverse colectomy	The middle colic vessels are divided flush at their origin.	The transverse colon is resected with both hepatic and splenic flexures, the transverse mesocolon, and the omentum.
Tumors of left colon	Left hemicolectomy	The inferior mesenteric vessels are divided at their origin.	The resection extends down to the rectum.
Tumors arising in sigmoid colon	Sigmoid colectomy	The sigmoid vessels are divided at their origin from the inferior mesenteric.	The sigmoid colon.
Tumors of upper 1/3 of rectum	Anterior restorative resection(**of Dixon**)	The inferior mesenteric is divided (or sometimes the left colic is spared)	The sigmoid colon, and the upper half of the rectum are excised. Bowel continuity is then restored.
Tumors of lower 1/3 of rectum	Abdomino perineal resection **(of Miles)**	The inferior mesenteric is divided (or sometimes the left colic is spared)	Similar to the above but the whole rectum is excised together with the anal canal, and a terminal colostomy is fashioned. The operation is called abdomino-perineal because part of it is done through the abdomen and the other through the perineum.
Tumors of middle 1/3 of rectum	Were used to be treated by abdomino-perineal resection and a colostomy like lower third tumors. Two factors made a change in this policy towards anterior restorative resection : 1. It is now realized that in the rectum 2cm safety margin below the tumor is compatible with radicality provided that the tumor is differentiated, thus leaving enough distal rectum for anastomosis. 2. The development of the circular staplers markedly facilitated this technically difficult procedure.		
Tumors of hepatic flexure	Extended right hemicolectomy	The ileocolic, right colic and the middle colic vessels are divided at their origin	The resection extends to the junction of the right two thirds and the left third of the transverse colon.

[B] Unobstructed inoperable (incurable) cases (whether inoperability is diagnosed before or during operation) :

1. Resectable tumor : Palliative resection of the colon cancer is offers the best palliation.

- Here there is no need for wide resection of the bowel nor its lymphatics and vessels.
- The operation is destined to obviate the risk of obstruction and bleeding.

2. If the tumor is irresectable : An operation is done just to avoid obstruction.

- Tumors of the right colon require : Side to side ileotransverse anastomosis.
- Tumors elsewhere in the colon : Proximal colostomy.

[C] Acute intestinal obstruction :

Urgent surgery is required after adequate preparation (see under intestinal obstruction) :

1. In cases of left colon :

- The best is colon resection (radical or palliative depending on operability), and colostomy.
- For the critically ill, proximal colostomy is done with later management as above.

2. In cases of right colon : (which are rare to obstruct) Primary anastomosis is done.

[D] Tumors of the rectum may show partial response to local radiotherapy.

- **Supplementary chemotherapy** with 5-fluorouracil (5-fu) may be useful for colon cancer.

Liver resection : requirements :

1. A completely resectable small colorectal cancer with no apparent residual tumor.
2. No extrahepatic metastases.
3. A solitary metastatic nodule or multiple metastases that are confined to one surgical lobe of the liver.

- **Liver resection is done in the same session of the colorectal operation, or later if the liver deposits appear in the course of the patient's follow up.**

Enumerate Small Intestinal Tumors

A. Benign tumors

- Adenoma
- Sub mucous lipoma
- Leiomyoma
- Peutz-jegher's syndrome

B. malignant tumors

- Adenocarcinoma
- Non Hodgkin's lymphoma
- Cracinoid tumor of small intestine

Rectal prolapse

Definition :

Protrusion of the rectum through the anus. When the mucosa only prolapses it is termed as partial (or mucosal) rectal prolapse. If the whole thickness of the rectal wall is everted to the outside, it is termed as complete rectal prolapse.

Etiology :

[A] Partial prolapse :

- Common in children due to :

1. Loss of the curve of secrum, the anus and rectum thus forming a vertical tube.
2. Loss of weight results in loss of the supporting pararectal and ischiorectal fat.
3. Straining at defecation e.g. due to prolonged diarrhea, or whooping cough.

- Partial rectal prolapse may occur in adults due to :

1. Advanced cases of hemorrhoids.
2. Continuous straining e.g. due to urethral stricture.
3. Atony of the sphincter mechanism in the elderly.
4. Injury to anal muscles during fistula operation.

[B] Complete prolapse :

The condition is commoner in the elderly, particularly females. In Egypt, young males may be more affected due to bilharziasis of the rectum.

Theories :

1. Failure of rectal support by the levators and pelvic fascia due to defective collagen maturation in the pelvic floor. Excessive stretching of these structures during labor may explain the association of prolapse with childbirth.
2. Rectal prolapse may represent a sliding type of hernia. As most patients have a deep rectovesical or rectovaginal extensions of the Pouch of Douglas (sac).
3. Abnormal mobility of the mesorectum leading to lack of fixation between the rectum and sacrum.
4. In Egypt bilharzial proctitis and colitis may be important predisposing factors as it leads to continuous tenesmus and straining at defecation especially if there are bilharzial ulcers and polyps.
5. Denervation of pelvic floor secondary to repeated childbirth.

Clinical features :

Symptom :

1. Prolapse :

- In early phases the prolapse appears on straining at defection & then reduces spontaneously.
- Later it may require manual repositioning.

2. Mucus discharge.

3. Bleeding from the congested mucosa.

4. A degree of fecal incontinence is a common symptom with complete rectal prolapse.

Signs :

- Examination in the squatting position is required if the prolapse does not appear in the lateral position.
- The anal sphincter tone is also assessed.

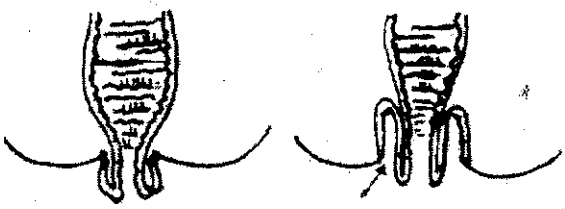
Items	Partial Prolapse	Complete Prolapse
Length of prolapsed	Less than 5cm	More than 5cm
Mucosal corrugations	Absent	Often present
Prolapse thickness	Formed of mucosa only	The rectal muscle thickness can be felt

Complications :

- Irreducibility, ulceration, and gangrene.

Differential diagnosis :

1. Prolapsing hemorrhoids.
2. Prolapsing polyp.
3. Prolapsing intussusception.



Prolapse *Intussusception*

Treatment :

1. In children :

- Surgery is rarely indicated as the condition resolves spontaneously.
- **Simple measures are helpful :**

1. Avoidance of constipation and straining.
2. Improving the nutritional status.
3. Manual reduction by the mother after defecation followed by strapping of the buttocks.
4. Submucous injection of 5% phenol in almond oil as for hemorrhoids is done in resistant cases.

2. In adults :

[A] Mucosal prolapse :

1. Submucous injection of 5% phenol in almond oil may be successful.
2. The prolapse is excised in a fashion similar to hemorrhoidectomy.

[B] Complete prolapse :

- Various surgical procedures include :

1. Rectopexy (Charles Well's operation) : Through an abdominal approach the rectum is mobilized, and is pulled upwards. A rectangular piece of prolene mesh is attached by sutures to the presacral fascia, and is then wrapped and sutured around the back and sides of the rectum.

2. Excision of the redundant rectum : The operation can be performed either through an abdominal or a perineal approach.

3. Delorme's operation : done through a transanal approach. The excess rectal mucosa above the dentate line is circumferentially excised. Sutures are used to approximate the upper edge of the remaining mucosa to that at the dentate line. The sutures are made to pass through the rectal muscle, plicating it.

4. Narrowing of the anus (Thiersch's operation) :

- Reserved for patients unable to stand a more major procedure.
- The prolapse is reduced, and a stainless steel wire or a thick monofilament suture is tunneled subcutaneously around the anus. The suture is then tied to fit snugly around the finger of the surgeon. Though the operation is it simplest, it provides the poorest results.

Anal Canal

Pilonidal sinus

There is a subcutaneous granulating cavity originally described as a nest (Latin-nidus) containing hair (Latin-pilus) and connected to the skin by midline openings (Latin-sinus).

Etiology :

- The disease typically affects young adult males with dense, dark, strong hair.
- **Two possible theories :**

1. Congenital theory : Infected dermoid cyst which present after puberty.
2. Acquired theory : More acceptable, Hairs from the head or back gravitate to the skin over the sacrum and coccyx. The hairs are drawn through the skin to accumulate in the subcutaneous tissue with debris and organisms.

- **Factors favour the acquired theory :**

1. Occurrence in other sites e.g. umbilicus, webs of fingers.
2. Microscopic picture shoes loose hairs with no hair follicles.
3. Tip of hairs is always directed inward.
4. Liability to recurrence.

Clinical features :

Symptoms : Patients may be asymptomatic. More usually, they present with discharge, or acute abscess formation. Pilonidal disease tends to recur if improper treated.
Signs : The discharging midline & lateral openings are seen, sometimes with loose hairs come out of them.
Differential diagnosis : Is from perianal abscess and anal fistula.

Treatment :

1. Pilonidal abscess : initially treated by incision and drainage of pus.
2. Pilonidal sinus : Different options are available :

- Opening the resulting wound is allowed to heal by secondary intention.
- Localized excision of the cavity and side tracks. The wound may be left open to granulate or is closed by sutures.
- **Wide wedge excision down to the sacrum should no longer be practiced as it is considered to be an unnecessarily extensive operation with very long healing time.**

Anal fissure

Longitudinal tear in the mucous membrane lining the anal canal as a result of :

1. Passage of hard stool leading to over distension of anal canal.
2. Dragging on the mucous membrane by polyp or fibrosed piles.
3. Injury by foreign body or speculum.
4. Crohn's disease.

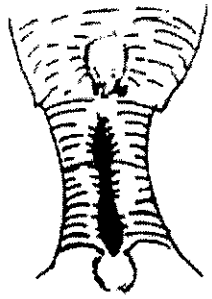
Pathology :

Site : 90% of fissures are posterior due to Y-shaped arrangement of the superficial part of external sphincter with absence of support posteriorly.

Character of acute fissure	Characters of chronic fissure
Simple superficial linear tear in long axis of anal canal.	Deep.
No fibrosis.	The edges are fibrosed and indurated.
Muscles of anal canal are spastic and not fibrosed (relax under anesthesia).	Organic fibrosis of the anal sphincter.
Pain leads to muscle spasm leading to more pain. Acute fissure usually does not heal spontaneously.	Associated sentinel pile at its lower end (due to infection and edema).

Complications : Abscess, fistula, fibrous contracture of anal sphincter leading to stricture formation.

Clinical picture :



1. **Pain :** sharp, agonizing, felt during and after defection.
2. **Discharge** which is scanty and serosanguinous.
3. **Bleeding is slight** i.e. streaks on stool.
4. **Constipation.**
5. **Reflex symptoms :** dysuria, dysmenorrhea.

On examination :

1. **Acute fissure** is seen with severe spasm of anal sphincter.
2. **Chronic fissure** is felt fibrosed with sentinel pile below.

D.D. of an anal fissure :

1. Fissures due to Crohn's disease. These are usually multiple, occur at any site and are not very painful. They are not indurated.
2. Tuberculous ulcers have undermined, cyanotic edges.
3. Early carcinoma of the anus.
4. Anal chancre presents as a painful ulcer.

D.D. of painful anal conditions :

1. Anal fissure.
2. Peri-anal suppuration.
3. Prolapsed strangulated piles.
4. Acute peri-anal hematoma.
5. Carcinoma of the anus.

Treatment :

[A] Acute fissure : (mainly conservative)

1. Relieve constipation by liquid paraffin & dieting.
2. Anesthetic ointment before defection.
3. Digital dilatation under anesthesia to relax the sphincter and prevent associated spasm.

- **If these measures fail internal sphincterotomy is done under general anesthesia.**

[B] Chronic fissure : (mainly surgical)

1. If the fissure is not very chronic :

- **A lateral internal sphincterotomy** operation is very successful.
- The internal sphincter is divided at the 3 o'clock position.
- Relief of the spasm of the internal sphincter will allow healing.
- The operation can be done by the closed or open method.

2. If the fissure is chronic with a sentinel pile :

- The best procedure is to do fissurectomy & posterior internal sphincterotomy.
- A triangular segment with its apex upwards and including the fissure, anal papilla and sentinel pile is excised.
- The operation should include internal sphincterotomy as well.

Hemorrhoids (Piles)

In Greek, hem means blood while rhoos means flowing. In Latin, pila means a ball. Piles may be internal or external according to its relation to the anal orifice.

Internal piles

They are varicosities of the tributaries of the superior hemorrhoidal veins.

Etiology : Primary or secondary

Secondary piles : Are due to :

1. Pregnancy due to raised intra-abdominal pressure and the relaxing effect of progesterone.
2. Pelvic tumor particularly carcinoma of the rectum may obstruct the tributaries of the superior rectal vein. Rectal carcinoma should be excluded in any patient above 40 with bleeding per rectum, even if there are evident hemorrhoids.

N.B.: Portal hypertension leads to anorectal varices.

[B] Primary piles : Occur without any organic cause.

Predisposing factors for the development of primary piles are :

1. Weakness in the wall of vein (hereditary).
2. Chronic constipation and straining at defecation.
3. Sphincteric relaxation due to loss of tone as in old age.
4. Anatomical factors :
   (a) Long column of venous blood sustained over the tributaries of superior hemorrhoidal vein as there are no valves in the portal circulation.
   (b) The veins are constricted as they pass through the muscle coat.
   (c) The veins lay in the lax submucosa and can be easily compressed by stool.

Pathology :

- Each pile consists of a dilated vein with an artery, connective tissue and covered with mucosa.
- They are arranged at 3-7-11 o'clock.

Clinical picture :

1. Bleeding : Which is bright red not mixed with stool which occurs at first with defecation but later at any time.

2. Prolapse or swelling :

- **1st degree piles :** no prolapse, patient may present by bleeding only. Diagnosed only by proctoscopy.
- **2nd degree piles :** prolapse only during defecation, reduced spontaneously at the end of defecation.
- **3rd degree piles :** prolapse during defecation and the patient has to manually reduce them.
- **4th degree piles :** permenant prolapse of piles.

3. Discharge & pruritis.

4. Pain is absent except in complications.

On examination : • P/R :

1. Piles are not felt but seen through proctoscopy except if complicated. In late cases, redundant folds of skin can be seen in the position of one or more of the three primary hemorrhoids. There may be daughter piles between the main 3 piles in elderly patients; sigmoidoscopy should be performed to exclude a rectal carcinoma.

2. Exclude rectal carcinoma.

Complications :

1. Bleeding which may be severe but commonly mild and repeated leading to anemia.
2. Strangulation of prolapsed pile by external sphincter leads to severe pain, edema, congestion and thrombosis.
3. Gangrene due to strangulation or thrombosis.
4. Ulceration and infection leading to peri-anal abscess rarely portal pyemia.
5. Sloughing of strangulated piles leading to secondary hemorrhage.
6. Fibrosis of thrombosed pile leads to formation of fibrous polyp.
7. Portal pyemia in infected piles.

Treatment : Treatment is either conservative, injection or surgical.

1. Primary piles :
- 1st & 2nd degrees : conservative and injection therapy is enough.
- 3rd & 4th degrees : treatment is mainly surgical.

2. Secondary pile : Treat the cause.

[1] Conservative treatment : Indicated in mild 1ry piles & 2ry piles.
- It consists of light non irritant diet, laxative, astringent ointment or suppository e.g. haemamelis.

[2] Injection sclerotherapy :
- **Indicated in :** Uncomplicated first and early second degree piles, secondary piles due to pregnancy and in recurrent piles after operation.
- **Method of injection :** 5-20% phenol in almond oil is injected submucous and extravenous opposite the pedicle of the pile 3-5 cc are injected and one pile each time is injected at one week interval.
- **Complications of injection :**

1. Sloughing due to injection in mucous membrane.
2. Submucous abscess.
3. Stricture of rectum due to excessive fibrosis.
4. Secondary hemorrhage.
5. Pain due to either injection too low in the subcutaneous space (very sensitive) or too deep in the internal sphincter.

[3] Rubber band ligation : (Barron's)
- Can be performed in 2nd & early 3rd degree hemorrhoids. It does not need anesthesia.
- The idea is to place a tight elastic rubber band around the pedicle of the hemorrhoid.
- Two hemorrhoids can be ligated at the same time.

[4] Cryosurgery : The application of liquid nitrogen (-196°C) causes coagulation necrosis of the piles.
- **Disadvantage :** The patient has prolonged mucous discharge.

[5] Photocoagulation : Infrared photocoagulation can give a tissue temperature of 100°C produces an area of coagulation necrosis.

[6] Surgical treatment : It is indicated in :
- 3rd & 4th degree piles.
- Profuse bleeding.
- Pulsating piles.
- Piles previously complicated.

Treatment of prolapsed strangulated hemorrhoids :
- It is commonly mentioned that strangulated hemorrhoids are a source of portal pyemia, although this is a rare occurrence. If the case is diagnosed early, surgical intervention under antibiotic is indicated.
- If the diagnosis is delayed, the tissues get friable and there may be secondary infection and so it is better to rely on conservative measures including rest in bed, antibiotics, analgesics, frequent warm baths, decongestive ointments and local compresses by lead acetate.
- Some surgeons perform maximal anal dilatation under anesthesia to relieve the sphincteric spasm.

External piles

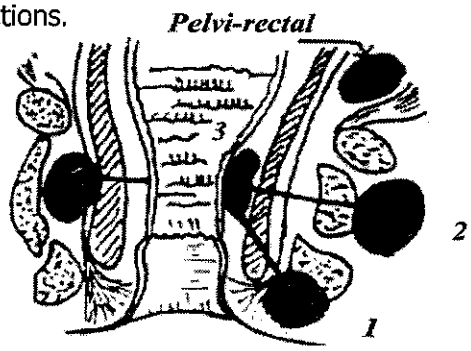
[1] Acute external piles	[2] Chronic external piles
• Sudden severe straining leads to rupture of tributaries of inferior hemorrhoidal veins with the formation of small perianal hematoma. • **Clinical Picture :** Bluish tender small swelling. • **Complication :** Ulceration, infection & fibrosis.	• Varicosity of inferior hemorrhoidal plexus in association with internal piles (intero-external piles). • Skin tags resulting from fibrosed hematoma. • Sentinel pile associated with anal fissure.
• **Treatment :** Cruciate incision evacuating the hematoma.	• **Treatment :** Excision of the tag in triangular manner.

Anorectal abscess

- **This is either primary or secondary :**

[A] Primary anorectal abscess :

- It is due to infection of the anal glands or secondary to skin infections.



Pathology :

- The anal glands exist in a plane between the internal and external anal sphincters and communicate by their ducts to the anal mucosa at the level of the dentate line.
- Infection of these glands by Gram-negative bacilli leads to the formation of an intersphincteric abscess which may spread :

1. Downwards : Perianal abscess.
2. Outwards : Ischiorectal abscess.
3. Inwards : Submucous abscess.
4. Upwards : High intersphincteric abscess.

- **In the majority of these abscesses, there is an inner opening in the anal canal and drainage of the abscess is usually followed by a fistula.**

[B] Secondary anorectal abscess : May develop in :

1. Inflammatory bowel disease as in Crohn's disease and ulcerative colitis.
2. Specific infections as tuberculosis.
3. Anorectal carcinoma.

Anorectal abscesses are classified anatomically into :

1. Perianal (60%) : The abscess lies subcutaneously adjacent to the anal orifice. The pain and constitutional symptoms are not marked.

2. Ischiorectal abscess (30%) : Marked constitutional manifestations. There is Large, indurated, tender swelling filling the ischiorectal space with throbbing pain and pitting edema. It may extend to involve the other side leading to horse shoe abscess.

3. Submucous abscess (5%) : The patient has severe pain and pyrexia but nothing is seen outside the anal verge. Per rectal examination reveals a tender boggy swelling.

4. Pelvirectal abscess (5%) : It is actually a pelvic abscess (see before).

Treatment :

- Urgent surgery **(don't wait for fluctuation)** under general anesthesia is performed.
- Perianal and ischiorectal abscesses are treated by making a cruciate incision over the abscess, opening all pockets of infection.
- A submucous abscess is drained by deroofing through the proctoscope.
- If the abscess is near the anal verge, the surgeon should look for an internal fistulous opening and if present, he should treat the patient as if having a fistula.

N.B.: The patient who had drainage of an anorectal abscess should be warned that he may develop a perianal fistula later on.

Anal fistula

Definition :

A Chronic granulating tract opening between the skin of the perianal region and the cavity of the anal canal or rectum .

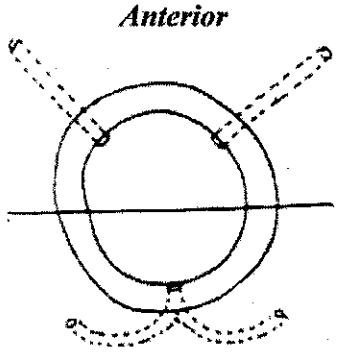
Etiology :

- It is common in males.
- It usually follows perianal suppuration.
- **Factors that help chronicity :**

1. The anal glands act as a reservoir for infection.
2. The presence of an internal opening allows recurrent activation of infection.
3. Underlying specific diseases such as Crohn's disease, tuberculosis, and ulcerative colitis.
4. Fecal material may act as a foreign body.

- **Care must be taken to exclude carcinoma of the anal canal or low rectum extending as a perianal fistula.**

Classification of anal fistulae :



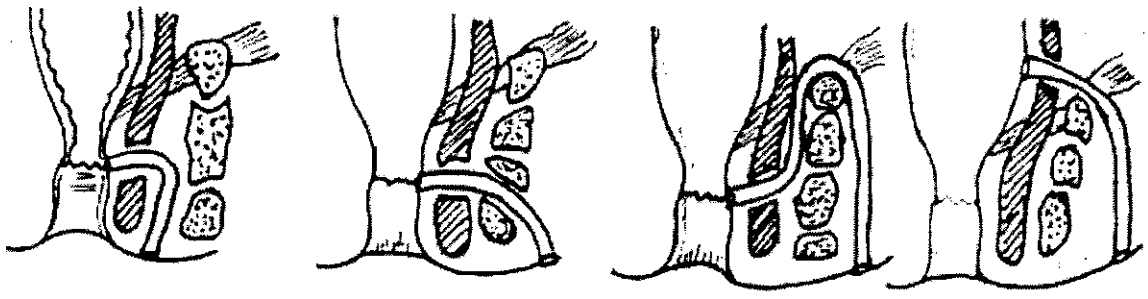
[A] Goodsall's rule : All fistulae behind the transverse anal line open by a common internal opening posteriorly in the middle line, while each external opening in the anterior aspect has its own internal opening. Posterior fistulae are curved **(horse-shoe)** while anterior fistulae are straight.

[B] Old classification :

1. Low anal fistulae : Have their internal opening below the anorectal ring.
2. High anal fistulae : Have their internal opening above anorectal ring in the rectum (usually iatrogenic).

[C] Recent classification : almost all anal fistulae have their opening at the level of the dentate line of the anal canal communicating to an intersphincteric abscess cavity.

1. Intersphincteric fistula : It passes between the internal and external sphincters. It may be associated with a high blind track.
2. Transsphincteric fistula : It passes through both the internal and the external sphincters. It may be complicated by additional blind tracks.
3. Suprasphincteric fistula : The track passes upwards along the intersphincteric plane, then laterally to above the anorectal ring to enter the ischiorectal fossa to the perianal skin.
4. Extrasphincteric fistula : The track passes outside the plane of the external anal sphincter. They usually have an intermediate communicating track passing through the external and internal sphincters to the original internal opening at the level of the dentate line.



Inter-sphincteric *Trans-sphincteric* *Supra-sphincteric* *Extra-sphincteric*

Clinical picture :

- History of an abscess which is followed by a persistent purulent discharge.
- One or more external opening are seen and palpated as tender indurated cord under the skin.

Investigation :

1. Proctoscopy may show the internal opening of the fistula.
2. Colonoscopy and/or barium enema may be needed to exclude an underlying specific cause.

Treatment :

[A] Simple fistula :

1. Fistulotomy (opening the fistulous track), curettage or cautery of the track and healing by secondary intention.
2. Fistulectomy (complete excision of the fistulous track) is not indicated because the magnitude of tissue loss associated with the procedure increases the risk of sphincter injury.

[B] Suprasphincteric fistula :

- **Fistulotomy will include division of the entire anal sphincter and will produce incontinence; so do staged fistulotomy :**

1. Initial stage : Is to deroof the lower part of the intersphincteric component. A suture is left in place through the rest of the track, snugly tied around the puborectalis and external sphincter.
2. Two weeks later : the rest of the track is opened guided by the suture.

[C] Extrasphincteric fistulae :

1. Treatment of the underlying cause.
2. Treatment usually includes proximal colostomy & staged fistulotomy.

Malignant tumors of the anal verge & anal canal

- The commonest type is **squamous cell carcinoma** arising from the anoderm. Less commonly adenocarcinoma arises above the dentate line.
- **Clinically** the patient presents by a lump, pain or discharge.
- **Metastases** may develop in the inguinal lymph nodes if the tumor is below the dentate line or to the para-aortic lymph nodes if the tumor is above the dentate line.

Treatment :

1. Carcinoma of the anal verge :

- Wide local excision with 2.5cm safety margin.
- If there are metastatic lymph nodes inguinal block dissection is performed.

2. Carcinoma of the anal canal : One of two lines may be performed :
(a) Abdomino-perineal resection with a terminal colostomy.
(b) Chemoradiation : A course of combination chemotherapy is followed by radiotherapy. The patient is examined after 4-6 weeks. If there is any evidence of residual tumor, abdomino-perineal resection is performed.

N.B. Colloid carcinoma may develop in the track of old standing perianal fistula.

Acute perianal hematoma (Thromboses external hemorrhoid)

- It is a very painful condition.
- Resulting from rupture of dilated anal vein & its spread secondary to straining at defection, coughing or lifting a heavy weight.
- It appears as tense, tender, bluish swelling covered by smooth shining skin.
- Untreated hematoma usually resolves but it may suppurate, ulcerate through skin with extrusion of the blood clot or fibrose → cutaneous tag.
- If patient presents early with severe pain → Hematoma is evacuated.

Fecal Incontinence

- It is the inability to retain the fecal contents at will.
- **Causes**:
  1. Damage of the anal **sphincter** mechanism:
     - Obstetrical trauma → complete perinea tear.
     - Surgical trauma e.g. surgery for high Anal Fistula
     - Accidental trauma (uncommon)
  2. Complete rectal **prolapse**:
     - Prolapsing rectum stretches the anal sphincters damaging them.
     - Excessive descent of perineum with straining → pudendal nerve traction neutopathy.
  3. **Idiopathic** fecal incontinence
  4. **Neurological** disease
     - Trauma or tumour affecting S2, 3, 4.
     - Diabetic autonomic neuropathy.
- **Assessment:**
  1. Clinical Assessment:
     - Degree of incontinence,
     - history of trauma or surgery
     - Neurological assessment
  2. Manometry to assess rectal function, length & strength of anal sphincters
  3. EMG → pudeudal never function.
  4. Defecography → Anorectal angle
  5. Trans-anal US & MRI → Localize site of sphincter damage

➧ ***Conservation treatment*** in mild cases.

1. Constipating agents & dietary manipulations to thicken stool
2. Anal sphincters & pelvic floor exercises.
3. Evacuating the bowel with a glycerine suppository in morning so as to avoid soiling later in day

➧ ***Surgical treatment***

1. Repair of divided sphincters
2. Rectopexy with restore continence to 50% in those having complete rectal propose.
3. Post-anal repair is indicated for idiopathic incontinence

Review Subjects

Abdominal injuries

Mechanism of injury :

1. Blunt injuries are due to :

- Direct blows to the abdomen.
- Sudden deceleration in motor car accidents.

2. Penetrating injuries are due to stabs or gunshots :

- When a bullet strikes soft tissues, shock waves spread out of the missile tract causing extensive damage to the surrounding tissues and even to areas far from the primary tract.

3. Blast injuries :

- The blast waves may lead to submucosal hemorrhages, mesenteric tears or perforations.
- Solid organs may be severely lacerated.

Clinical picture :

[A] History of trauma.

[B] General : There may be :

1. Picture of hypovolemic shock or the patient may be toxic (due to peritonitis).
2. Look for associated injuries especially **associated chest injuries.**

[C] Local : Trauma to the abdomen may cause :

I- Abdominal wall injury (only) :

1. Pain at the site of trauma with stable general condition.
2. Tenderness localized to the site of injury, but there are no spreading physical signs.
3. Clinical picture of the wound.

II- Intra-abdominal injury : (in addition to the above)

1. Injury to a solid organ or major blood vessels leads to internal hemorrhage.
2. Injury to a hollow viscus leads to peritonitis.

3. Specific signs according to the injured organ e.g.:

(a) Injury to the biliary radicles : jaundice due to absorption of the bile pigments from the leaking bile.
(b) In case of liver injury : there may be hematobilia.
(c) In case of rupture spleen : (see rupture spleen).
(d) In case of renal injury : there may be hematuria.
(e) In case of urinary bladder injury : there may be retention of urine, suprapubic pain & tenderness.

Investigations & management :

• If the clinical picture of the patient indicates frank peritonitis or internal hemorrhage exploration should be performed without losing time in doing unnecessary investigations.

Emergency investigations in abdominal injuries are indicated for :

1. Suspicion of organ injury with equivocal signs.
2. Unreliable abdominal examination because patient is unconscious e.g. head trauma, drug or alcohol intoxication.
3. Unexplained hypotension that may be caused by blood loss.

Emergency investigations include :

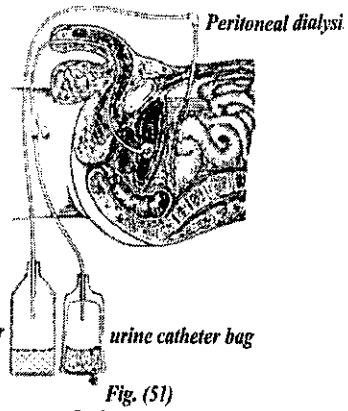


Fig. (51)
Peritoneal lavage in blunt abdominal trauma

I- Serial blood picture and hematocrit value :

- Decreasing hemoglobin and hematocrit value denote blood loss.
- Polymorphnuclear leucocytosis points to peritonitis.

II- Radiological :

[A] plain chest and abdominal X-ray(only in stable patients): may reveal:

Facture ribs,foreign body,or air under diaphragm

[B] Focused Abdominal sonography of trauma(FAST) may reveal :

a) Can be performed at the bed side.
b) 85-95% accuracy in detection of intaabdominal fluid , blood ,and perisplenic
c) Or perihepatic hematoma.

Disadvantages:

- Operator dependent
- Not sensitive in bowel perforation.

[c] CT abdominal scan (only in stable patients):

- Very accurate in detecting solid organ injuries,retroperitoneal and diaphragmatic injuries.
- Not sensitive in bowel and acute pancreatic injuries.

III- Diagnostic Peritoneal lavage (DPL) :

- This is the **investigation of choice** for cases of blunt abdominal trauma with equivocal signs.
- Under local anesthesia, a small catheter is inserted through a small midline incision and 20 ml/kg of sterile saline is instilled by a drip set.
- The patient is rolled laterally in both directions and then the fluid is permitted to drain back.
- **Positive result if :**

1. Frank blood, blood stained pink fluid & bile stained fluid indicate an intra-abdominal injury.
2. A red cell count more than 100.000/mm3, a white blood cell count higher than 500/mm3, or elevated amylase.

IV- Diagnostic laparoscopy.

Decision :

1. If investigations reveal an intra-abdominal injury, laparotomy is done.
2. If investigations reveal no evidence of viscus injury, the patient should still be kept under observation in hospital for two days. Development of new signs may necessitate a delayed laparotomy.

Treatment :

[1] ABCDE.

[2] Antibiotics.

[3] Abdominal exploration :

Indications for urgent abdominal exploration in cases of abdominal trauma without losing time in investigations :

1. General and local clinical manifestations of intra-abdominal bleeding.
2. General & local clinical manifestations of peritonitis.
3. Stab wounds with a protruding viscus.
4. All missile injuries.

- A large exploratory abdominal incision is done (either midline or right paramedian incision).
- Thorough exploration of whole abdominal organs for possible injuries.
- **Dealing with each injury accordingly e.g.:**

1. Injury of spleen, liver, kidney & intestine (see before).
2. Injury of the gall bladder → cholecystectomy.
3. Injury of the CBD → repair on T-tube.

[4] Treatment of associated injuries : Take care of associated chest injuries

Acute abdomen

Definition :

It is a term applied to any abdominal pain requiring urgent relief.

Clinical picture :

- **Type of patient differs according to the cause.**

[1] Pain : Usually the most important symptom.

Types of abdominal pain :

1. Visceral pain	2. Somatic pain
Due to distension of a hollow viscus.	Due to irritation of the parietal peritoneum.
Site : it is felt in the segment of the abdominal wall having the same nerve supply of the affected organ and it is vague ill-defined pain.	**Site :** it is well localized over the affected organ. e.g. pain of acute appendicitis (discuss).

- **Sudden onset of pain denotes** hemorrhage, perforation or torsion.
- **Gradual onset of pain denotes** inflammation.

[2] Vomiting : Usually occurs once or twice after the onset of pain.

[3] Bowels :

- Constipation is the rule in most cases of acute abdomen.
- Tenesmus may be present in pelvic appendicitis.
- Mesenteric thrombosis may be accompanied by bleeding per rectum.

[4] Urinary problems and menstrual disturbances : Should be asked about.

Classification of acute abdomen :

[A] According to the mode of presentation

1. Colics e.g.: Intestinal, appendicular & biliary.
2. Inflammations e.g.: Appendicitis, cholecystitis & pancreatitis.
3. Perforations : Perforated appendix & peptic ulcer.
4. Intestinal obstruction.
5. Internal hemorrhage.
6. Urological causes : Calculi & inflammations.
7. Gynecological causes : (see acute appendicitis)
8. Medical causes of acute abdomen : (see acute appendicitis)

[B] According to the site

Upper abdominal :

1. Perforated peptic ulcer.
2. Acute cholecystitis & biliary colic.
3. Acute pancreatitis.
4. Leaking abdominal aortic aneurysm.
5. Mesenteric vascular occlusion.
6. Acute myocardial infarction.

Mid abdominal :

1. Intestinal obstruction.
2. Mesenteric vascular occlusion.

Right lower abdomen :

1. Acute appendicitis.
2. Colitis.
3. Meckel's diverticulitis.
4. Right ureteric colic.
5. Crohn's disease.
6. Mesenteric adenitis.

Left lower abdomen :

1. Colonic diverticulitis.
2. Colitis.
3. Left ureteric colic.

Pelvic :

1. Pelvic inflammatory disease.
2. Complicated ovarian cyst.
3. Pelvic appendicitis.
4. Ectopic pregnancy.
5. Proctitis.
6. Cystitis.
7. Prostatitis.

Abdominal & back pains :

1. Posterior duodenal ulcer penetrating the pancreas.
2. Acute cholecystitis & biliary colic.
3. Acute pancreatitis.
4. Leaking abdominal aortic aneurysm.
5. Spine disease with radicular pain that radiates forwards.
6. Renal & ureteric colic.

Investigations :

In the majority of cases accurate clinical examination supplemented by few tests can confirm the diagnosis. The following are some useful investigations :

[1] Laboratory :

- Hemoglobin & hematocrit.
- TLC.
- Serum amylase.
- Electrolytes.

[2] Plain x-ray of the chest in the erect position :

- May show free gas under the copula of the diaphragm in perforation of a viscus.
- Basal pneumonia will be detected.

[3] Plain x-ray of the abdomen may reveal :

- Calculi of the urinary tract.
- Distended loops of bowel or fluid levels in intestinal obstruction.

[4] Abdominal ultrasound can diagnose the following :

- Acute calcular cholecystitis.
- Acute pancreatitis.
- Leaking aortic aneurysm.
- Distended pelvicalyceal system in cases where ureteric stones cause colic.
- Gynecological disorders diagnosed by pelvic ultrasound.

[5] CT scan of the abdomen can diagnose the following :

- Acute pancreatitis.
- Retroperitoneal hemorrhage.

Upper GI bleeding

Definitions :

Upper GI hemorrhage is usually due to lesions above the ligament of Treitz (end of duodenum).
It manifests by hematemesis and/or melena.

- **Hematemesis :** Vomiting of blood which will be of a coffee ground material if it is small amount or bright red blood if it is of a large volume.
- **Melena :** is passage of black tarry stools.
- **Hematochezia :** Occasionally upper GI bleeding is so massive and the GI transit is so fast to the extent that it manifests by fresh bleeding per rectum.
- **Occult GI bleeding :** Minor trickling of blood from any part of the gut that neither manifest in vomitus nor in change of color of stools.

Hematemesis

Definition :

- Vomiting of blood which will be of a coffee ground material if it is small amount or bright red blood if it is of a large volume.
- It is usually due to lesions above the ligament of Treitz (end of duodenum).

Etiology :

[A] Esophageal causes :

1. Esophageal varices. 2. Esophagitis. 3. Esophageal cancer. 4. Mallory-Weiss syndrome.

[B] Gastroduodenal :

1. Peptic ulcer (gastric & duodenal). 2. Fundal varices. 3. Gastritis.
4. Multiple gastric erosions & acute hemorrhagic gastritis. 5. Gastric carcinoma or benign tumors.

[C] General causes :

1. Swallowed sharp objects. 2. Hemorrhagic diseases. 3. Hematobilia. 4. Uremia.

[D] False hematemesis e.g. vomiting of swallowed blood from epistaxis.

Bleeding esophageal varices is the commonest cause of hematemesis in Egypt. It should be stressed that a patient with esophageal varices may bleed from another lesion (e.g. gastric erosions or peptic ulcer) which are not rare associations.

Other common causes :

1. Multiple gastric erosions.
2. Acute hemorrhagic gastritis.
3. Chronic peptic ulcer.

D.D.:

1. Is the vomited material really blood, or some reddish material ?

2. Exclude hemoptysis (coughing blood).

Items	Hematemesis	Hemoptysis
1. History	GIT troubles	Chest troubles
2. With	Vomiting	Cough
3. Color	Dark red	Bright red
4. Content	Food	Froth
5. Signs	Abdominal	Chest
6. Followed by	Melena	Blood tinged sputum

3. Exclude false hematemesis.

Management of patients with active bleeding :

Life saving resuscitative measures should be initiated immediately and are then followed by diagnosis and definitive treatment.

I- Estimation of severity of bleeding and resuscitation.
II- Localization of the site and cause of bleeding.
III- Treatment of specific lesions.

I- Estimation of severity of bleeding and resuscitation :

1. Admit to hospital. Severe bleeding cases require ICU management.
2. Initial then repeated clinical and hematocrit assessment.
3. Insert two peripheral venous lines and withdraw blood for cross-matching and blood tests.
4. Insert a Foley catheter. Urine output is the best monitor of tissue perfusion.
5. A central venous line is needed for monitoring of severe cases.
6. IV sodium containing fluids e.g. Ringer's lactate are started until blood is available.
7. A naso-gastric tube is inserted for all cases.
8. If the patient is irritable, give 5-10 mg morphia IV to sedate him. Morphine, however, is contraindicated in cases of liver insufficiency.
9. Correct coagulopathy by fresh frozen plasma and by giving the missing factors
10. A major cause of morbidity and mortality is aspiration of blood. To prevent this complication in patients with altered mental status, endotracheal intubation should be considered.

II- Localization of the site and cause of bleeding :

History:

- Previous attacks and their management, Hepatitis and Shistosomiasis.
- Medications, particularly NSAIDs & Peptic ulcer symptoms.
- Bleeding tendency.

Examination:

- Stigmata of cirrhosis & Surgical scars.
- Tenderness.

Laboratory tests:

- Hemoglobin percent and hematocrit value will show evidence of hemodilution after 3 hrs
- Liver function tests will be disturbed in patients with cirrhosis and esophageal varices.
- Blood urea and creatinine.
- Exclude causes of generalized bleeding tendency by the coagulation tests.

Fibreoptic endoscopy:

- Should be performed as early as possible once the patient has been resuscitated.
- It is done under a mild sedative as diazepam.

Barium radiography:

- Is loosing favor because it is less accurate than endoscopy.

Angiography:

- In difficult situations where radiology or endoscopy fails to diagnose the lesion that causes bleeding, it may be necessary to resort to celiac angiography to pinpoint the source of the bleeding.
- Angiography needs to be performed during active bleeding.

III- Treatment of specific lesions :

- Bleeding esophageal varices
- Bleeding peptic ulcers, erosive gastritis and multiple gastric erosions.

Bleeding per rectum

Bleeding per rectum may present as one of three forms :
1. Occult bleeding per rectum.
2. Melena.
3. Fresh bleeding per rectum (Hematochezia).

Fresh Bleeding Per Rectum

Hematochezia is fresh bleeding per rectum.

Etiology :

[A] The commonest causes are : lesions of the anal canal as hemorrhoids and fissure.
[B] Generalized causes : as thrombocytopenia or leukemia.
[C] Colorectal causes :
1. Drug intake as anticoagulants, NSAIDs or steroids can cause colonic bleeding.
2. Congenital lesions. Familial polyposis coli.
3. Inflammatory lesions :
   (a) Acute bacillary dysentery. (b) Bilharzial colitis.
   (c) Amebic dysentery. (d) Ulcerative colitis.
4. Vascular lesions as ischemic colitis.
5. Diverticular disease of the colon.
6. Neoplastic lesions. Benign and malignant neoplasms of the colon and rectum.
7. Angiomatous malformation of the colon.

N.B.:
- **Hemorrhoidal bleeding is the commonest cause.**
- **Massive bleeding per rectum in adults :**

1. Diverticula.
2. UC.
3. Ischemic colitis.
4. Angiodysplasia.
5. Massive bleeding from upper GIT.

Clinical picture :
1. Anemia & hypovolemia. 2. C/P of the cause.

Investigations :
- **Check that the patient does not have upper gastrointestinal bleeding by passing a nasogastric tube or by upper endoscopy.**

1. Exclude the causes of generalized bleeding tendency.
2. Stools examination may reveal bilharzial ova or amebiasis.
3. Proctoscopy, Sigmoidoscopy.
4. Colonoscopy : in patients with massive colonic bleeding, the blood will obscure the field.
5. Isotope scans : The patient's own RBC's are tagged with 99mTc and then injected intravenously. Abdominal scanning by a gamma camera can identify the site of bleeding.
6. Angiography : will localize the site of bleeding provided that the rate of bleeding is 0.2-0.5 ml or more per minute. Injection of vasoconstrictors or gel foam through the angiography catheter, which may be therapeutic.
7. Barium enema is only justified as an elective investigation in case of chronic blood loss.
8. Laparotomy : If all the previous investigations failed to pinpoint the site of bleeding.

Treatment :

- **Resuscitation.**

- **In the majority of cases, the bleeding will stop spontaneously** and the surgeon has the time to diagnose and treat the patient electively.

- **If massive bleeding continues :**

1. Therapeutic embolization.
2. If colonoscopy visualizes an era of vascular malformation (angiodysplasia), bleeding can be stopped by diathermy or laser.
3. Urgent surgical intervention :

(a) If the source of bleeding could be localized preoperatively, segmental resection of the colon would be performed.

(b) If not, total colectomy may be indicated.

N.B.:

- **Commonest causes of massive fresh bleeding per rectum :**

1. Diverticular disease.
2. Angiodysplasia of the colon.

- **Commonest causes of fresh bleeding per rectum :**

1. Hemorrhoids.
2. Carcinoma of the colon and rectum.
3. Diverticular disease.
4. Angiodysplasia of the colon.

Occult bleeding per rectum

- This means the passage of a small quantity of blood per rectum that cannot be detected by the gross appearance of the stools.
- To examine stools for occult blood, special tests are performed depending on the peroxidase activity of hemoglobin **e.g.guaiac test.**
- A positive test denotes the presence of a source of blood, loss of about 20ml/day, e.g., chronic peptic ulcer or malignancy of the gastrointestinal tract.

Melena

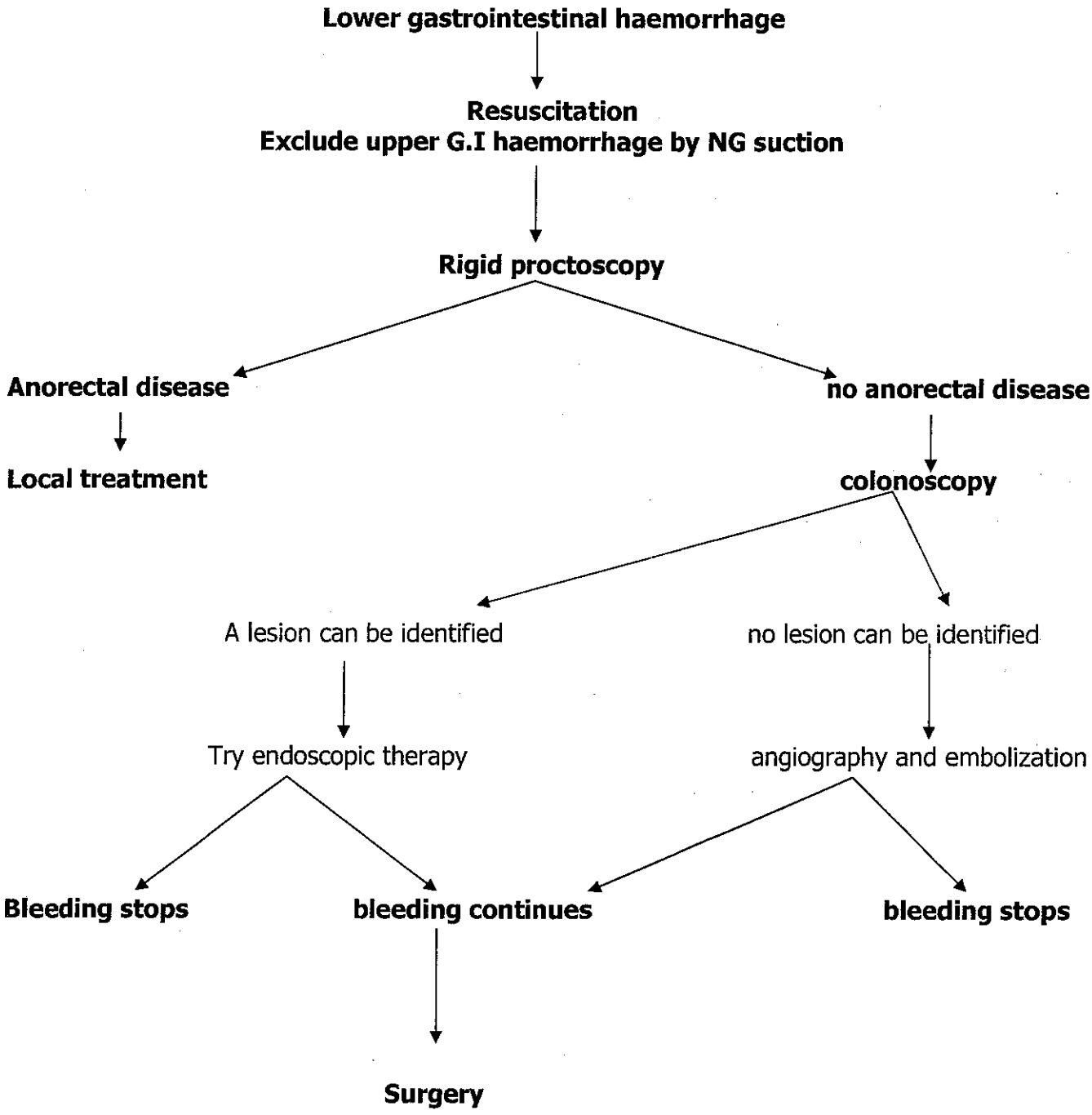
Definition : the passage of black tarry stools.

Etiology :

- Bleeding from lesions above the ileocecal valve usually presents as melena, but if the bleeding is massive or if the patient has diarrhea, the patient may present with fresh bleeding per rectum.

- Causes of melena include all causes of hematemesis due to lesions of the small bowel as neoplasms, bleeding Meckel's diverticulum, Crohn's disease, mesenteric vascular occlusion and bleeding typhoid ulcers.

Algorithm for management of lower gastrointestinal haemorrhage



Causes of abdominal distention or vomiting following an abdominal operation

1. Acute gastric dilatation.
2. Paralytic ileus.
3. Intra-abdominal abscess, e.g. subphrenic or pelvic.
4. Peritonitis due to a leaking anastomosis or perforation of a hollow organ.
5. Adhesive intestinal obstruction.
6. Medical causes as uremia or diabetic keto-acidosis.

Causes of post-operative pyrexia

1. In the first 24 hours, mild pyrexia may occur as a reaction to the stress of the operation.
2. In the first few days the commonest cause of pyrexia is respiratory complications.
3. Wound infection.
4. Deep vein thrombosis.
5. Thrombophlebitis at the site of a cannula.
6. Urinary tract infection especially in the presence of a catheter.
7. Intra-abdominal abscess.

Large abdominal cysts

1. Pancreatic pseudocyst (upper abdomen).
2. Mesenteric cyst (mid abdomen with characteristic mobility).
3. Ovarian cyst (pelvi-abdominal).
4. Hydronephrosis (extends to the renal angle).

Hernia

Diseases of the Umbilicus

1. Umbilical fistula :

(a) Fecal fistula : Is either congenital from a patent vitello-intestinal duct, traumatic, inflammatory from tuberculosis of small intestine or malignant from carcinoma of the transverse colon ulcerating through the umbilicus.

(b) Urinary fistula : Is either congenital from patent urachus or is rarely acquired.

(c) Biliary fistula : Is due to perforation of an inflamed gall bladder.

2. Umbilical sinus : Discharging pus from abdominal wall abscess or umbilical infection. Pilonidal sinus of the umbilicus is rare.

3. Umbilical stone.

4. Enteroteratoma (Umbilical polyp).

5. Umbilical granuloma.

6. Umbilical hernia.

7. Tumors of umbilicus :

(a) Squamous cell carcinoma.

(b) Secondary carcinoma nodules.

Desmoid tumor

It is a locally malignant fibrosarcoma. Arises from the anterior abdominal wall muscles. It may occur on top of scars or incisions.

Treatment : Is by excision with a safety margin of at least one inch and reconstruction of the defect.

Complications of abdominal incisions

1. Hematoma : May be due to bleeding tendency, more commonly due to careless surgical hemostasis. It causes painful indurated wound. If of large size, it should be evacuated to avoid secondary infection.

2. Infection.

3. Wound disruption (burst abdomen) : May cause mortality.

4. Incisional hernia : Deep layers disruption of wound during the immediate or early post-operative period.

5. Desmoid tumor.

N.B: rectus sheath hematoma:

May be secondary to trauma or iatrogenic(following abdominal operations) with injury of superior or inferior epigastric vessels.**treatment:** conservative,aspiration or surgical evacuation and ligation of bleeding vessels.

Burst abdomen

Predisposing factors: see incisional hernia.

Types:

- **Partial**. The deep layers burst but the skin is intact.
- **Complete**
- If the intestine prolapses out of the wound, it is called **evisceration**.
- If the intestine is retained inside the abdomen, it is called **dehiscence**.

Clinical features:

- A warning sign to the occurrence of burst is "**the red sign**" where a serosanguinous discharge soaks the dressing.
- The burst usually occurs on **6th** to the **8th** day post-operatively.
- Symptoms of intestinal obstruction may be present.

Treatment: is by urgent surgical closure.

- Cover the prolapsed bowel by a sterile dressing.
- Insert a nasogastric tube and start IV infusion.
- The abdominal wall is closed as one layer by through-and-through sutures using strong non-absorbable suture material, e.g., polypropylene. These are called "**retention sutures**".

Hernia

Definition :

Protrusion of a viscus or part of a viscus (usually within a sac) through a defect in its containing cavity

Types :

[A] External abdominal hernia : It is protrusion of a viscus or part of a peritoneal sac through a defect in the abdominal wall.

1. Inguinal hernia. 2. Incisional hernia. 3. Femoral hernia. 4. Epigastric hernia.
5. Paraumbical hernia. 6. Umbilical hernia. 7. Divarication of recti. 8. Rare types.

[B] Internal hernia :

1. Diaphragmatic hernia. 2. Paraduodenal hernia. 3. Paracecal hernia. 4. Paracolic hernia.
5. Hernia through a defect in the broad ligament of the uterus.
6. Hernia through the foramen of Winslow.

Etiology :

1. Congenital factor : Due to the presence of a preformed sac (as congenital inguinal hernia).

2. Acquired :

(a) Any condition that raises the intra-abdominal pressure : As whooping cough, chronic cough, pregnancy, straining at micturition (stricture or prostatic obstruction), straining at defecation (constipation) and powerful muscular effort.

(b) Weakness of abdominal wall : As a result of obesity, pregnancy, old age and previous operation.

Anatomy :

The hernia consists of a sac, contents and coverings.

[A] Sac : Consists of a diverticulum of peritoneum which is divided into : neck, body and fundus. Neck is usually narrow and well defined.

[B] Contents : May be any of abdominal viscera except the pancreas e.g.:

1. Omentum (Omentocele)	2. Bowel (Enterocele)
Doughy.	Soft.
On reduction → no gurgle sensation.	On reduction → give gurgle sensation.
Percussion → dull.	Percussion → resonant.
Easy reducible at first but difficult at the end.	Difficult reduction at first but easy at the end.

3. A part of the circumference of the intestine = Richter's hernia.
4. Meckel's diverticulum = Littre's hernia.
5. Two loops of the bowel = Maydl's hernia (W hernia)

[C] Coverings : Are derived from the layers of abdominal wall Through which the sac passes.

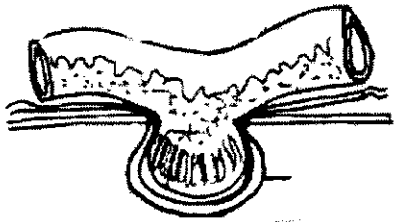


Fig. (52)
Richter's hernia

N.B.:

Richter's hernia :

1. Common in femoral hernia due to small sac.
2. Strangulation occurs without intestinal obstruction (gives picture of gastroenteritis).

Clinical picture :

- Patient complains of an intermittent swelling at the site of a hernial opening.
- The swelling increases in size on standing, straining or cough and is reducible on lying down.

On Examination : Swelling in an anatomical site of hernia

1. The swelling increases in size in all directions when the patient cough ; **Expansible impulse.**
2. The swelling is reduced in the abdomen ; **Reducibility.**
3. The hernial orifice can be palpated. Its obliteration with the thumb after reduction of the hernia prevents its descent.
4. Consistency : See table.
5. Percussion : See table.
6. Transillumination : Opaque.
7. A non complicated hernia is not hot, not red, not tender, not tense and expansile impulse on cough is always present.

Investigations :

1. Routine pre-operative :
2. Investigations to detect precipitating factors e.g. Plain X-ray chest, abdominal US and I.V.U.

Treatment :

1. Correct any predisposing factor.

2. Truss : Is a palliative treatment and is indicated when the patient is unfit or hernia is very liable to recurrence. Truss can only be applied to a reducible hernia after reduction.

3. Operative treatment : Is the only method of cure :

[A] Herniotomy (better called herniectomy) : Removal of the sac as far as the neck.
- By itself it is sufficient for the treatment of inguinal hernia in children.

[B] Herniorrhaphy : It consists of excision of the sac and reconstruction of the defect by local tissues.

[C] Herniplasty : It is indicated in :
- Very wide hernial defects (can not be closed by sutures).
- Very poor muscles (can not protect the defect).
- Repeated recurrence.

Complications of Hernia :

1. Irreducibility. 2. Obstruction. 3. Inflammation. 4. Strangulation.
5. Rupture due to trauma. 6. Hydrocele of hernial sac.
7. Torsion of the omentum. 8. Interference with normal activity in huge hernia.
9. Complications of treatment : (a) Truss. (b) Taxis. (c) Surgery.

Irreducible Hernia

The contents cannot be returned to the abdomen.

Causes :

1. Adhesion between sac and contents. 2. Over crowding of contents within the sac.
3. Sliding hernia. 4. Narrow neck.

Clinical Picture : The hernia can not be reduced by patient or surgeon, but the hernia is not hot, not tender, nor tense and impulse on cough is still present.
Complications : Obstruction and strangulation.
Treatment : Operation is always indicated (not an emergency). No truss can be used.

Obstructed Hernia

A hernia containing intestine the lumen of which is obstructed either from without by band of adhesion or from within the lumen by a mass of stool (this type is termed **incarcerated hernia**).

Clinical picture :

1. Patient complains of repeated attacks of **abdominal colic, vomiting, abdominal distension** and **absolute constipation.**
2. The hernia is irreducible, soft, not hot, not red and not tense.

Treatment : Distinction between obstruction and strangulation in hernias may be difficult, thus, it is safe to treat it as strangulation and early surgery should be performed.

Inflammation

Causes :

1. Inflammation of contents as appendix.
2. Trauma by attempts at taxis or pressure of truss.

Clinical picture :

1. General manifestations of inflammation.
2. The hernia is painful, hot, red and tender and impulse on cough can be felt.

Treatment : Operation is essential as strangulation can be missed.

Strangulated Hernia

Definition :

A hernia in which the blood supply of the contents is impaired leading to gangrene follows (may occur within 5-6 hours).

Incidence :

- Strangulated hernia is the commonest cause (with adhesive obstruction) of acute intestinal obstruction.
- It varies according to the type of hernia. It is estimated to be about 2-4 % in inguinal, 25-30% in femoral, 15-20% in paraumbilical and 3-5% in incisional hernias.
- Although the incidence of strangulation is higher in femoral hernias, yet, strangulated inguinal hernias account for more than 50% of all strangulated external hernias.
- Strangulation can occur at any age and it is commoner after prolonged use of a truss.

Predisposing Factors :

1. Irreducibility, obstruction and inflammation.
2. Sudden expulsion of new contents.
3. Repeated attempts at reduction producing edema.
4. The hernia may be strangulated at the time of its first descent, especially in infants.

Pathology :

[A] The constricting agent may be :

1. A resistant structure outside the sac as the superficial or deep inguinal ring or the lacunar ligament.
2. The neck of the sac.
3. Bands of adhesions within the sac.

[B] Contents :

Interference with the venous pressure occurs first (venous pressure being low)
→ congestion & edema → further rise of pressure → impairment of the arterial supply

- Mucosa Sloughs within three hours and bleeds in the lumen.
- Within six hours the whole thickness of the intestinal wall is affected, and exudes serosanguinous fluid in the peritoneum.
- The intestine becomes gangrenous and soon perforates producing peritonitis.

• **Gangrene** (and then perforation) appears at the :
1. Ring of constriction
2. Apex of the loop → peritonitis.

Clinical picture :

[A] General :

• **Picture of acute intestinal obstruction** with colicky abdominal pains, vomiting, distention and constipation.
• The patient is **dehydrated** and has **hypovolemic shock.**
• With the onset of gangrene, peritonitis occurs and produces paralytic ileus and the colic stops **(a bad sign).**
• In late stage, there is **picture of both hypovolemic and septic shock.**

N.B.: No obstruction occurs with omentocele, Richter's hernia or Littre's hernia.

[B] Local :

• Severe pain.
• Sudden enlargement.
• Irreducibility.
• **No impulse on cough.**
• The hernia becomes **tense** and **tender** due to extravasation of fluids and peritoneal irritation.

N.B.:
1. In Maydl's hernia, gangrene affects first the loop which lies in the abdomen.
For this reason during operation for strangulation all loops are explored.
The local tenderness over the hernia of this type is not marked during clinical examination.

2. Strangulated Richter's hernia may be spontaneously reduced and the gangrenous part of the intestine may be overlooked at the operation.

D.D. of strangulated hernia :

1. From other complications.
2. From other causes of acute swellings in the anatomical region of the hernia.
3. From other causes of acute intestinal obstruction.

Treatment of strangulated hernia :

I- Resuscitation : Anti shock measure, fluid replacement, blood transfusion and gastric suction.

II- Taxis : i.e. reduction of strangulated hernia by manipulation.
This is done only within the first 6 hours after the onset and only in a previously reducible hernia.

Complications of taxis of strangulated hernia (better avoided) :

1. Shock.
2. Rupture of intestine.
3. Reduction en masse i.e. the sac with its strangulated contents is reduced as a whole into the abdomen.
4. Peritonitis due to squeezing of harmful fluid of the sac into the peritoneal cavity.
5. Volvulus of reduced bowel.
6. Reduction en bisac : contents are reduced from main sac into secondary sac.

III- Operation : Should be done as soon as possible :

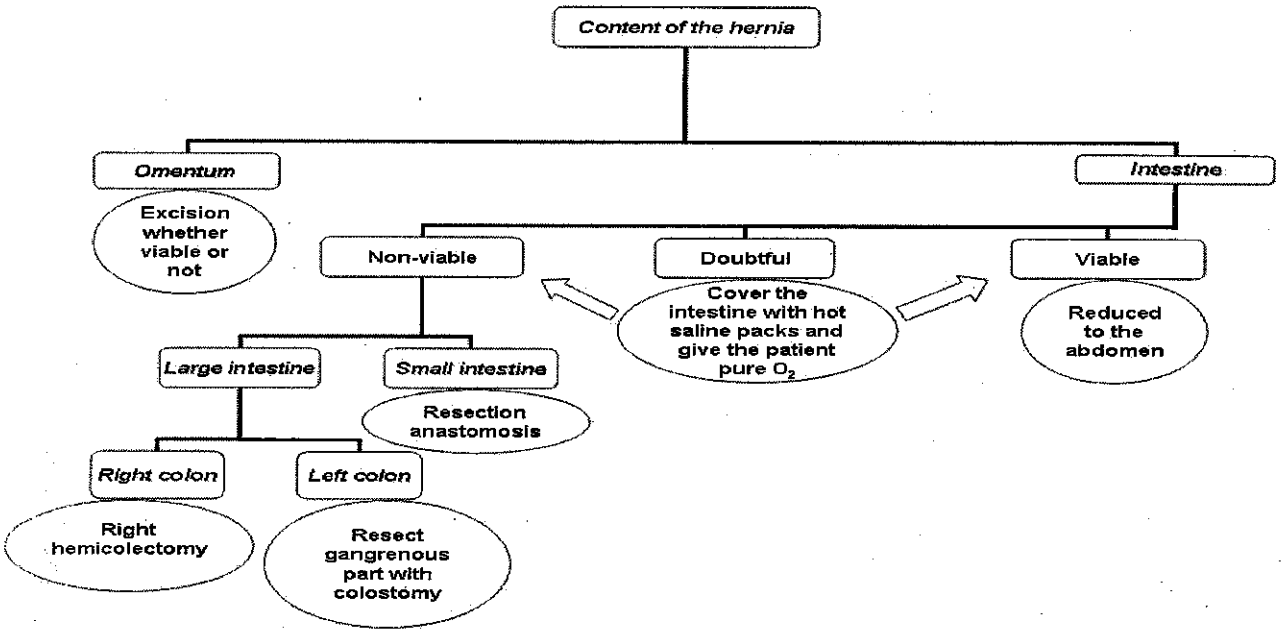
1. Long incision is done reaching down to the fundus of the sac.

2. Fundus of sac is exposed first and is opened before the neck. The fluid in the sac is drained. This avoids escape of the harmful fluid into the peritoneal cavity and avoids peritonitis.

3. Constriction at the neck is relieved.

4. Pull on the loop of intestine present in the sac to reach loops inside the abdomen and examine for strangulated W-hernia **(Maydl's hernia)** in which the strangulated loop of intestine is inside the peritoneal cavity while the loops of intestine in the sac appear normal.

5. Contents of the hernia are dealt with as follow :



Items	Viable intestine	Non-viable intestine
1. Peristalsis.	Present.	Absent.
2. Peritoneal lusture.	Present.	Absent.
3. Mesenteric Pulse.	Present.	Absent.
4. Color	Pink or dark red.	Green, brown or black.
5. Tone	Present (recoil after release ofpressure).	Absent.
6. Fluid in the sac	Yellowish, odorless	Dark, offensive.

Intraoperative tests to detect viability :

1. Doppler ultrasound.

2. Fluroscein test : Inject 1 gm fluroscein I.V., and then inspect the bowel under U.V. rays. If the bowel has good blood supply it will flurescence.

3. Oximeter.

6. Repair of the hernia defect.

7. Subcutaneous drains are usually needed.

Hydrocele of the hernial sac

- **This occurs in narrow necked sacs** if contents return to abdomen and fail to descend into sac again.
- **The neck of the sac** becomes occluded by omentum and serous fluid collects in the sac.
- **Clinically** there is a cystic swelling in the upper part of the cord.

Inguinal hernia

Hernia which traverses the inguinal canal to emerge at the external inguinal ring.

N.B:protective mechanisms of the canal

- The shutter mechanism: contraction (straightening) of conjoint tendon when straining.
- Obliquity of the canal.
- Support of weak points of the canal e.g. conjoint tendon lies behind external ring.
- Bulging mechanism of cremasteric muscle by contraction during straining.
- Increased obliquity of the canal during straining.

(1) Oblique Inguinal Hernia

It is the most common of all hernias. It may be due to the presence of a preformed sac which is part of the patent procesus vaginalis. This is congenital in origin. In old age most of the cases are acquired and are due to muscular weakness and raised intra abdominal pressure.

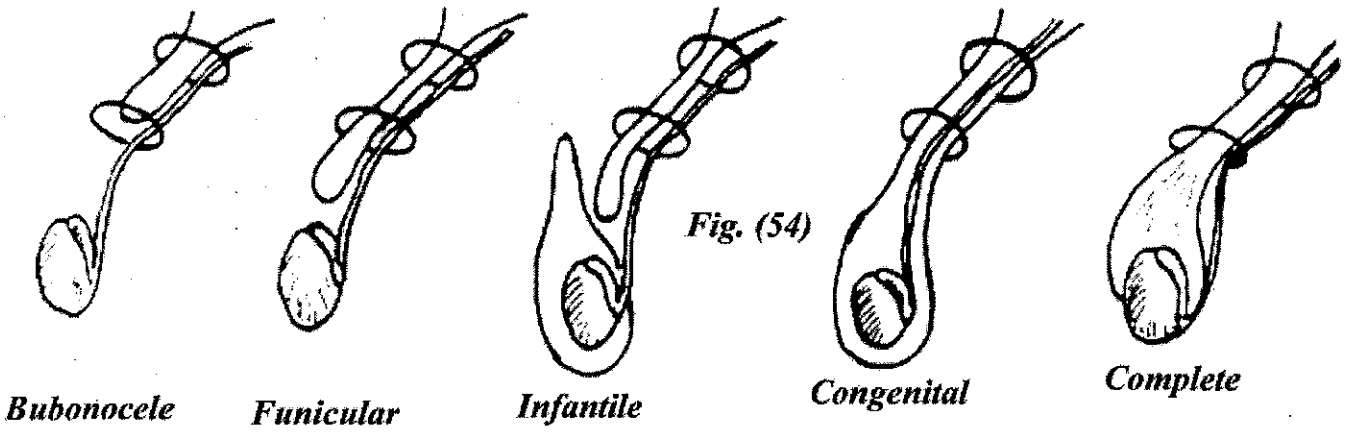


Fig. (54)

Anatomical types :

[A] Congenital type :

The whole procesus vaginalis is patent and thus the general peritoneal cavity is connected to the tunica vaginalis. Though called congenital it may not appear until adult life.

[B] Infantile type :

In which the tunica vaginalis extends up wards towards the inguinal canal. At operation the tunica is liable to be opened in mistake for true sac which will be found behind it i.e. two sacs are found within the cord:
A) Hernial sac.
B) The extension of tunica vaginalis upwards in the cord.

[C] Adult type :

1. Bubonocele : Hernia limited to inguinal canal and stops just distal to superficial inguinal ring.
2. Funicular hernia : Hernia passes with the cord and stops just above the epididymis.
3. Complete inguinal hernia (scrotal hernia) :
Long standing hernia which has reached the bottom of the scrotum. The sac is not continuous with the tunica vaginalis and the testis can be felt separate from the sac (D.D. from congenital inguinal hernia).

Anatomy :

1. The defect : Stretched deep ring.
2. Sac : Escapes from deep ring and lies always inside the cord within the coverings, being anterolateral to the vas and vessels.
3. Contents : See before.
4. Coverings : Skin, superficial fascia, external spermatic fascia from external oblique), cremasteric muscle (from internal oblique), internal spermatic fascia (from transversalis fascia).

Clinical picture : More in males, any age.
Symptoms : Pain, intermittent swelling either inguinal or inguino-scrotal which is reducible.
Signs : As general :

- Direction of reduction is upward laterally and backwards.
- Internal ring test : see clinical notes.
- External ring test : see clinical notes.
- **Transillumination in inguinal hernia :** Opaque (an inguinal hernia may be translucent in infancy or early childhood, but never in adult).

Treatment : See before.

N.B.: Inguinal hernia in children :

1. Herniotomy once diagnosed (for fear of complications).
2. If strangulated : Try taxis if presented early, followed by herniotomy after 48 hours.
3. Bilateral hernias can be operated upon in same setting.

The congenital type of OIH is characterized by :

1. Sudden appearance.
2. Descends into the bottom of the scrotum at once (other types reach the bottom of the scrotum in several years).
3. Completely surrounds the testis and can not be separated from it.
4. Become strangulated at the moment of descent.
5. Found mostly in young children.

(2) Direct Inguinal Hernia

Incidence : 20% of inguinal hernias.

Etiology : Always acquired due to weakness of muscles with associated chronic straining.

Pathology :

The sac enters the inguinal canal behind the spermatic cord.
The sac is small; hemispherical with a wide neck.
The coverings are the skin, external spermatic fascia, conjoined tendon, transversalis fascia and extra peritoneal fat.

Clinical picture and differentiation from the oblique type :

Items	Indirect inguinal hernia	Direct Inguinal hernia
1. Age	Any age.	Usually old.
2. Sex	More in male.	Always in males.
3. Side	Unilateral or bilateral.	Usually bilateral.
4. Shape	Pyriform.	Hemispherical.
5. Descent	Forward medially & downward.	Directly forward.
6. Reduction	Upward, laterally & backward.	Directly backward.
7. Site	Inguinal or inguinoscrotal.	Only inguinal.
8. Internal ring test	(Hernia does not descend).	(Hernia will descend).
9. Defect (seen at operation)	Deep ring, lateral to inferior epigastric vessels.	Hasselbach's traingle, medial to inferior epigastric vessels.
10. Complications	Common.	Rare.

Treatment : See before.

Femoral hernia

Definition :

A hernia which leaves the abdomen through the femoral ring into the femoral canal. It represents 20% of hernias in females and 5% of hernias in men.

Etiology : More in females due to :

1. Larger female pelvis.
2. Less developed ileo psoas muscle.
3. Repeated pregnancies.
4. Pelvic tilt.

Pathology :

- The hernial sac passes down the femoral canal vertically till it reaches the saphenous opening. It is prevented from further downwards descent. The sharp superior edge of the saphenous opening turns the hernial sac superiorly.
- So the hernia passes forwards pushing before it the cribriform fascia, then curves upwards and laterally, towards anterior superior iliac spine.

Clinical picture :

- Patient presents with a small reducible **(frequently irreducible)** swelling at the upper medial part of the thigh below the inguinal ligament.
- The swelling is not tense or tender and an impulse on cough is present.
- The swelling is below & lateral to the pubic tubercle (D.D. inguinal hernia which is above & medial).

Treatment : See operative notes.

Differential diagnosis :

[A] Reducible femoral hernia	[B] Irreducible fem
1. Inguinal hernia.	1. Irreducible inguinal he
2. Saphena varix.	2. Lipoma.
3. Aneurysm of the femoral artery.	3. Inguinal lymphadenopathy.
4. Psoas abscess.	4. Ilioposas bursa.

Comparison between Inguinal hernia & Femoral hernia

Items	Inguinal hernia	Femoral hernia
Site	Above & medial to pubic tubercle.	Below & lateral to pubic tubercle.
External & internal ring tests	+ ve	- ve
Descent	Forwards, medially & downwards.	Downwards, forwards & upwards.
Reduction	Upwards, laterally & backwards.	Downwards, backwards & upwards.

Complications :

- Frequently femoral hernia becomes irreducible and commonly it gets strangulated. This is accounted for by the narrow unyielding femoral ring and sharp border of lacunar ligament.
- Richter's hernia is not uncommon.

Rare types of femoral hernia

1. Cloquet's hernia: The sac lies under the fascia covering the pectineus muscle.Strangulation is likely.The sac may coexist with the usual type of femoral hernia.

2. Laugier's femoral hernia Is a hernia through a gap in gimbernat's ligament. The diagnosis is based on the unusual medial position of a small femoral hernia. Nearly always the hernia is strangulated.

3. Narath's femoral hernia: the hernia lies behind the femoral vessels,occurs only in patients with congenital dislocation of hip.

Umbilical hernia

I- Congenital Umbilical Hernia (Exomphalos)

1. Exomphalos minor :

A small defect (less than 5cm) is present at the umbilicus through which a small peritoneal sac protrudes. The contents are usually intestine. The coverings are a thin layer of Wharton's jelly and a layer of amniotic membrane.

Treatment : The contents are reduced the sac is excised and the defect is repaired in layers.

2. Exomphalos major :

There is a large defect (more than 5cm) in the center of the abdominal wall usually above the umbilical cord. The contents may include many viscera or part of the liver. The covering is only a layer of amniotic membrane. There is a danger of rupture of the sac followed by peritonitis.

Treatment : Urgent operation

- The skin on either sides of the defect is undermined and approximated together over the sac and sutured.
- Release incisions in the flanks are needed.
- After several months the peritoneum and muscles can be approximated and closed in layers.

II- Infantile umbilical hernia

- The neck of the sac is wide and the coverings are extraperitoneal fat and umbilical scar.

Treatment :

1. Correction of the cause of straining if any.
2. Strapping is better avoided as in most cases the defect closes spontaneously within 2 years.
3. Operation is indicated when the defect is more than 2 fingers wide or when the hernia persists after the age of 2 years. The defect in linea alba is closed with few stitches of non-absorbable suture material as prolene.

III- Adult umbilical hernia (Paraumbilical hernia)

Etiology :

It is more common in middle aged females, especially in obese multiparous women usually, paraumbilical and not umbilical hernia.

Surgical pathology :

- **Paraumbilical hernia is more common above the umbilicus** because the linea alba is thin and broader above than below the umbilicus.
- **The sac has a narrow neck.**
- **Adhesions** inside the sac are very common especially at the fundus.
- **Complications** as strangulation and irreducibility are very common due to the narrow neck, sharp edge and adhesions inside the sac.

Treatment : See operative notes.

- **Why in adults it is a para umbilical and not true umbilical hernia ?**

As the umbilical scar is the strongest part of the linea alba which is wider and thinner above the umbilicus than below.

Epigastric hernia

It is herniation of extra peritoneal fat through a small defect in the linea alba. It may be single or multiple.

Anatomy :

- **The commonest site** is midway between umbilicus and xiphoid process.
- The hernia commences as a protrusion of extraperitoneal fat only **(fatty hernia of linea alba).**
- If the protrusion enlarges, it drags a pouch of peritoneum after it **(true epigastric hernia).**
- **The neck of the hernia is narrow,** consequently, either the sac is empty or it contains a small portion of greater omentum.
- **Coverings :** Skin and subcutaneous tissue.

Clinical picture :

- Small swelling which may be irreducible simulating a small lipoma.
- It may be symptomless. Sometimes severe epigastric pain with nausea and vomiting from traction of herniated omentum on the stomach (D.D. peptic ulcer).

Treatment :

- If there is pain, the surgeon should be sure that it is not due to an underlying disease e.g. peptic ulcer or gall stones.
- Operation is performed by excision of hernial sac and closure of the defect in the linea alba.
- If the defect is large, it is repaired by Mayo's operation.

Divarication of recti

This is separation of the recti due to stretching of linea alba by a chronically raised intra-abdominal pressure.

Clinical Picture :

- It is common in middle aged females due to repeated pregnancies and in young males due to Egyptian splenomegaly.
- When the abdomen is relaxed, nothing is visible, but on raising the shoulders from the bed, the linea alba bulges as a longitudinal ridge between the two recti, the fingers can be dipped into the abdomen between the two recti.

Treatment :

- An abdominal belt is satisfactory in symptomless cases.
- If surgery is necessary **Keel operation** is the most suitable. The operation is discussed under the treatment of incisional hernia.

Incisional hernia

Hernia developing after abdominal incision.

Etiology :

[A] Pre-operative causes :

Obesity, malignant disease, jaundice, anemia and hypoproteinemia.

[B] Operative causes :

1. Operations for peritonitis, pancreas with leakage of enzymes and intestinal obstruction.
2. Incision : Muscle cutting, damage to nerve or blood supply to muscles.

[C] Post-operative causes :

1. Post-operative cough, chest complications and distension.
2. Post-operative wound infection.

Clinical picture :

- A swelling through the scar of an operation which is reducible, with an impulse on cough.
- It may contain omentum or intestine.

Treatment : See operative notes.

N.B.:• Direct hernia protrudes through the Hasselbach's Δ which is bounded by :

1. Medially → Rectus muscles.
2. Laterally → Inferior epigastric vessels.
3. Below → Inguinal ligament.
4. Floor → fascia transversalis.

- **The hernia (contra distinct to oblique type)** : Lies posterior to the spermatic cord.
- **Hasselbach's triangle is divided into medial and lateral halves by lateral umbilical ligament so the direct hernia leaves the triangle through its :**

1. Lateral part → lateral direct hernia.
2. Medial part → medial direct hernia (more liable to strangulation).

Pantaloon hernia (Dual or saddle - bag) :

- Here there are 2 sacs, one sac being med. & the other lateral to inferior epigastric vessels.
- One of the sacs may be overlooked at the time of operations.

Sliding Hernia :

Definition : Hernia where a viscus forms a part of the wall instead of contents.

- The commonest viscus implied is the urinary bladder.
- The colon may be implicated on the right side (cecum) or left side (sigmoid).

Common in left side.

When to suspect sliding bladder ?

Hernia is dull on percussion, fluctuant & by pressure on it urine is evacuated or the patient experiences a strong desire to pass urine, partially irreducible, double micturition, and huge long standing hernia.

Treatment of sliding hernia :

A U-Shaped incision is made in the peritoneum at a distance of about 2 cm from the bowel. The gap in the posterior wall of the sac is closed by suture. The bowel is replaced now within the abdominal cavity. The neck of the sac is closed by a purse string suture.

Rare External Hernia

1. Spigelian Hernia :

A hernial sac pass through the apponeurosis of transversus abdominis (Spigelian fascia) which forms the lateral Border of Rectus sheath commonly at the level of arcuate line.

2. Lumbar hernia : May be :

(a) Inferior lumbar hernia : passes through the inferior lumbar triangle of Petit which is bounded below by iliac crest, lateral by the external oblique and medially by latissimus dorsi.

(b) Superior lumbar hernia : passes through the superior lumbar triangle bounded by 12th rib above, medially by sacrospinalis and laterally by posterior border of internal oblique.

3. Perineal hernia : Passes through the pelvic floor.

4. Obturator hernia : Passes through the obturator canal.

5. Gluteal hernia : Passes through the greater sciatic foramen.

6. Phantom hernia : due to muscular paralysis or weakness e.g. Malgaigne's bulges.

7.interstitial hernia: the sac passes between anterior abdominal wall muscles.diagnosis may be difficult and may need CT.

8. Amyand's hernia: inguinal hernia with strangulated appendix.

D.D. of inguinoscrotal swelling :	D.D. of Inguinal Hernia :
1. Inguinal hernia. 2. Infantile or encysted hydrocele. 3. Undescended testis. 4. Cord swelling : (a) Vas : T.B. (b) Veins : Bilharzial, varicocele. (c) Lymphatics : Filariasis.	1. Direct from indirect. 2. Femoral hernia. 3. Hydrocele. 4. Undescended testis. 5. Lipoma of the cord.

Testis & Scrotum

Congenital anomalies

1. Imperfect descent of the testis :
(a) Incomplete descent of the testis.
(b) Ectopic testis.
© Retractile testis.
2. Testicular agenesis or hypoplasia (rare).
3. Supernumerary testis (rare).
4. Anteversion : (a) Anterior anteversion : The epididymis lies in front of the testis.
(b) Lateral anteversion : The epididymis lies lateral to the testis.
(c) Transverse anteversion.

Incompletely descended testis (Undescended testis)

Incidence :

- It affects about 1% of all males (higher in premature infants).
- Commoner on the **right** side.
- Bilateral in about 20% of cases (cryptorchidism)

Etiology :

[A] Mechanical factors :

1. Large size of the testis and epididymis.
2. Shortness of the spermatic vessels or the vas deferens.
3. Adhesions fixing the testis in higher places.
4. Rupture of the gubernaculum.
5. Short mesorchium

[B] Hormonal deficiency : Due to decreased levels of gonadotrophic hormones.

Pathology :

- **The testis may arrest anywhere along the normal line of descent :**
  1. In the abdomen (intra-abdominal).
  2. In the inguinal canal.
  3. At the external ring.
  4. At the neck of the scrotum.
- **After 2 years,** there is retardation of the development of the testis due to the higher temperature.
- **At puberty,** irreversible destructive changes in the germinal epithelium occur and in bilateral cases, sterility is usually the rule.
- **The hormonal function** of the testis remains almost normal.
  - ➡ The secondary sexual characters develop normally even in bilateral cases
- **There is associated oblique inguinal hernia** in more than 90% of cases.

Complications :

1. Sterility in bilateral cases.
2. Increased incidence of trauma especially in intracanalicular type.
3. Torsion of the spermatic cord.
4. Complications of associated hernia.
5. Epididymo-orchitis.
6. Psychiatric disturbance.
7. The most serious complication is the higher incidence of malignancy.
   - ➡ **The increased risk of testicular malignancy especially in intra-abdominal type is genetically determined and may affect the patient even after successful orchiopexy. However, orchiopexy should help early diagnosis.**

Clinical picture :

1. **The ipsilateral side** of the scrotum is empty and underdeveloped.
2. **The testis may be felt** in an abnormal site.
3. **An associated hernia** may be present.
4. **Hermaphroditism** should be excluded in bilateral cases.

Differential diagnosis :

1. Ectopic testis.
2. Retractile testis.
3. Testicular agenesis.
4. Testicular atrophy e.g. mumps orchitis.

Investigations :

1. Hormonal assay (LH and testosterone hormone) : To exclude cases of anorchia in bilateral cases.
2. Ultrasonography and CT scan : Can help to localize the site of the testis when it is not felt.
3. Laparoscopy : For intra-abdominal testis.

Treatment : Surgical treatment

Orchiopexy :

- **Best age** between 1-2 years.
- **Incision :** Inguinal incision opening the inguinal canal.
- **Associated hernial sac,** is removed i.e. herniotomy is done.
- **Cord elongated** by dissecting it high up and cutting any anchoring bands. Inferior epigastric artery may be divided to gain more length for the cord.
- **The most popular method** for fixation of the testicle is to place it in a **Dartos pouch** created between the skin of the scrotum and the Dartos muscle.

Orchiectomy :

- Removal of the undescended testis if it is atrophied or diseased (Torsion or malignancy) provided that the other testis is normal.

Management of difficult cases :

- The difficulty usually arises from a short testicular artery.
- This can be dealt with by one of the following methods :

1. Staged orchiopexy : The testis is brought down in more than one stage.
2. Fowler Stevens operation : High division of the testicular vessels provided that the anastomosis between the testicular artery and the artery of the vas is intact.
3. Microvascular technique.

Ectopic testis

The testis passes out of the external inguinal ring, and then it passes to a subcutaneous ectopic position.

Etiology : It may be due to the pull of an accessory gubernaculum.

Pathology : The hormonal and the spermatogenic functions are normal.

- The testis may be present in :

1. The inguinal region.
2. In the perineum.
3. At the root of the penis.
4. In the femoral triangle.

Ectopic inguinal	Undescended inguinal
On straining, the testis becomes more apparent.	On straining, it becomes less apparent.
Testis can be pushed medially but NOT laterally.	The reverse.
The spermatic cord can be felt medial to the testis.	Cannot.

Treatment : Replacement of the testis in the scrotal bed. Fixation may not be necessary.

Retractile Testis

- Occurs in young children due to exaggerated cremasteric reflex.
- It can be differentiated from incompletely descended testis by :

1. The scrotum is well developed.
2. The testis can be pushed into scrotum by sliding the fingers down the inguinal canal especially if the child squats and draws the thigh to the abdomen, or if the child is examined during sleep in a warm room.

Treatment : No treatment is required. Just reassurance.

N.B.: D.D. of empty scrotum :

1. Retractile testis (the commonest).
2. Undescended testis.
3. Ectopic.
4. Surgically removed.
5. Agenesis.
6. Atrophic testis may not be palpable.

- When both testes are in the abdomen & are consequently impalpable, the condition is known as cryptorchidism (crypt = hidden place).
- Undescended testis is commoner on right side (50%) than on the left side (30%) as right testis descends later (for the same reason, right obligue inguinal hernia is more common).

Torsion of the spermatic cord (Torsion of the testis)

Etiology :

- **Predisposing causes include :**

1. Imperfect descent of the testis (incomplete or ectopic).
2. Long mesorchium.
3. Capacious tunica vaginalis.
4. Spirally attached cremasteric muscle.
5. Transversely lying testis with the cord attached to its upper pole (Anteversion).
6. Sinus of the epididymis leading to rotation of the testis itself.

- **The exciting cause** is usually a minor trauma or straining at defecation.

Pathology : The testis rotates from outside inwards :

- Gangrene occurs within 8-12 hours if the condition is not treated.

Clinical picture :

1. Sudden severe agonizing pain in the scrotum, groin and the lower abdomen. **It may be associated with shock & vomiting.**

2. On examination : The scrotum is swollen with irreducible acutely tender swelling. The overlying skin is edematous and red. The testis is higher than normal and sometimes the twists are felt in the cord.

Differential Diagnosis :

[1] Torsion of spermatic cord & Acute epididmo-orchitis :

Torsion of spermatic cord	Acute epididmo-orchitis
Age : usually in adolescents and children	Adults and elderly
There may be **history** of mild trauma	History of a predisposing factor e.g. urinary tract infection may be present
Temperature : Normal or slightly elevated	Elevated
Elevation of the scrotum does not alleviate the pain	Some relief is obtained by elevation
Urgent urine examination is free	May show UTI
Ultrasound and duplex examination of the testicular vessels reveal obstruction of the blood flow	Patent testicular vessels

N.B.: If the diagnosis of torsion of the testicle is suspected clinically, exploration is indicated even if it proves negative. Any delay may lead to loss of the testicle.

[2] Torsion of an undescended testicle has to be differentiated from a strangulated inguinal hernia.

Investigations :

1. Urgent Doppler : To detect obstruction of the flow in the vessels.
2. Urgent Duplex scan : To detect the thrombus in the vessels as well.
3. Urine examination.

Treatment : Urgent operation

1. In early cases : Untwist the cord "fix the testis to the scrotum to prevent recurrence" eversion of the tunica to prevent hydrocele formation. The opposite testis should also be fixed at the same time as the anatomical variation responsible is likely to be bilateral.
2. In late cases : where the testis is gangrenous the treatment is orochiectomy.

N.B.: Causes of gangrene of testis :

1. Torsion testis.
2. Acute filarial funiculao-epidiymitis, gangrenous type.
3. Neglected strangulated inguinal hernia.

Inflammatory conditions of the testis & spermatic cord

1- Acute Epididymo-orchitis

Etiology : Infection is :

- Either hematogenous through blood stream.
- Or descending, spreading along the vas in cases of urethritis, cystitis and prostatitis.

Clinical picture :

- Fever, malaise and rigors.
- Scrotal pain referred to the corresponding iliac fossa.
- The epididymis and testis are swollen hot and tender with secondary hydrocele.
- Urine analysis may show pus cells.

Treatment :

- Antibiotics
- Elevation of the scrotum.
- Rest in bed.
- If pus forms immediate incision is indicated.

2- Chronic Epididymo-orchitis

It may follow an acute attack or chronic form the start and inflammation is localized to epididymis.

Treatment : Urinary antiseptic and antibiotics.

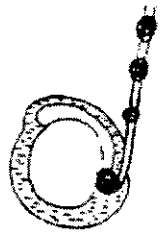
3- T.B. Epididymitis

Infection is either blood borne, or spreads along the vas from prostate or seminal vesicles.

Pathology :

- Infection starts in the head of epididymis in blood borne infection and in its tail in descending infection along the vas.
- Infection starts as a hard nodule which gradually increases in size with appearance of other nodules.

Clinical picture :



- Young males more affected.
- Slight ache in the testis with a small swelling.

On examination :

- Hard tender nodule in the tail or head of epididymis and later the whole epididymis becomes enlarged.
- In later stages cold abscess develops and opens through the skin of scrotum as a discharging sinus at the lower and posterior part of the scrotum.
- The vas is thickened and beaded.
- Secondary hydrocele.
- P/R may show thickened seminal vesicles or hard nodules in the prostate.
- **T.B. sinus occurs anteriorly in anteverted testis.**

Treatment :

1. Conservative : Anti-T.B. treatment.

2. Operative : If no response to medical treatment or in the presence of a discharging sinus.

(a) Epididymo-vasectomy : Is done if the disease is confined to the epididymis, removing the epididymis and the vas and leaving the testicle.

(b) Orchidectomy : When the testis is invaded.

4- Bilharziasis of the cord

The disease affects the lower end of the cord and epididymis but the testis and tunica are not affected.

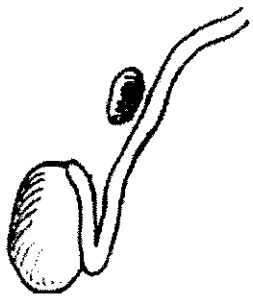
- There are 3 types :

1. Massive type (rare) : The cord, epididymis and testis are embedded in a firm fibrous mass which ends at the neck of scrotum.

2. Nodular form (commonest) : The terminal part of the cord or head of epididymis is the seat of a fibrous mass which is firm with some few nodules above it.

3. Granular form : Multiple firm yellowish grey bilharziomata infiltrate the cord

Clinical Picture :



- Swelling in the cord accompanied by dragging sensation.
- On examination a firm painless swelling in the cord which never extends into the inguinal canal.
- The vas is felt separate as the bilharzial affection is restricted to the veins where the ova were deposit.

Treatment :

- Anti-bilharzial treatment.
- Excision of the mass may be done in the massive and the nodular type.

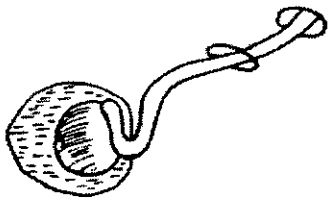
5- Filariasis of the cord

[A] Acute endemic funiculo epididymitis :

- Microfilaria leads to lymphatic obstruction. Secondary streptococcal infection leads to lymphangitis and thrombophlebitis of lymphatics and veins of the cord which is affected up to the internal ring.
- Inflammation may proceed to suppuration, extensive thrombosis or sloughing.

Clinical Picture :

- Sudden onset of severe pain with fever rigors and malaise.
- The cord and epididymis are swollen and tender.
- The cord is matted up to the internal abdominal ring.
- Secondary hydrocele may be felt.



Filariasis of the cord

Treatment :

1. Antibiotics. 2. Hetrazan. 3. Incision if pus forms.

[B] Chronic funiculo-Epididymitis :

1. Nodular form : (rare) rounded firm mass in the lower part of the cord or head of epididymis which is attached to the vas. Cord is thickened and may be matted.

2. Diffuse form : (common) The whole cord is thickened and matted extending from epididymis to internal abdominal ring. Epididymis is thickened and small hydrocele may be felt.

Treatment : Nodular form may need surgical excision

[C] Lymphocele : Soft mass of dilated lymphatics in the cord.

D.D. : from varicocele, lymphocele does not vary in size with posture.

[D] Diffuse hydrocele of the cord : Diffuse edema of the cord due to lymphatic obstruction

Syphilis

Syphilitic orchitis may be :

1. Diffuse interstitial fibrosis : The disease is usually bilateral and may lead to sterility.

2. Solitary Gumma of the testis : There is painless, hard, globular swelling with loss of testicular sensation. Later, syphilitic ulcer occurs which is situated anteriorly.

3. Bilateral orchitis : Usually occurs in congenital syphilis.

Neoplasms of the testis

Incidence :

- 99% of testicular neoplasms are malignant.
- They constitute 1-2% of malignant tumors in males.

Etiology :

1. Imperfectly descended testis, especially the intra-abdominal variety. The incidence of malignancy in undescended testicle is 15 times as the normal population.
2. Surgery for undescended testicle does not prevent the increased susceptibility to malignancy.
3. Systemic administration of estrogens may lead to Leydig cell tumor.

Classification of testicular tumors :

1. Germ cell tumors :

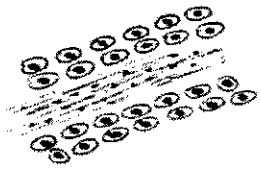
- Seminoma (40%).
- Teratoma (32%).
- Combined seminoma and teratoma (14%).

2. Interstitial tumors (1.5%) :

- Leydig cell tumor.
- Sertoli cell tumor.

3. Lymphoma (7%).

4. Other tumors (5.5%).

Items	I- Seminoma	II- Teratoma
Age	Older age	Younger (20-35)
Origin	Carcinoma from the seminefrous tubules.	Arises from the embryonic totipotent cells giving rise to ectoderm, mesoderm & endoderm.
Pathology : **1. N.E.**	• The enlarged testis is firm & smooth. • Non-capsulated.	• It may reach a big size. • Non-encapsulated, variable in consistency & the surface is slightly irregular.
2. Cut section	• Homogenous. • Occasionally fibrous septa give it a lobulated appearance. • In rapidly growing tumors areas of necrosis & hemorrhage are sometimes present, in this cases the consistency is variable.	• Variable in consistency & appearance with areas of hemorrhage & necrosis.
3. Histologically	• **Cells :** Rounded with large rounded nuclei. Arranged in columns and sheets separated by a fine fibrous stroma. • **Stromal & tumor infiltration** by lymphocytes suggests a good host reaction & tends to improve the prognosis.  *Fig. (58)*	**1. Teratoma differentiated (T.D.) :** dermoid cyst. **2. Malignant teratoma intermediate M.T.I. :** The most common. **3. Malignant teratoma anaplastica (M.T.A.).** **4. Malignant teratoma trophoblastica (M.T.T) :** was known as choriocarcinoma the most malignant associated with production of chorionic gonadotrophin.
4. Growth	Slow	Rapid
5. Malignancy	Lower	Higher
6. Spread	Mainly by lymph to para aortic L.N.	Mainly by blood to lung
Irradiation	Very radiosensitive	Radioresistant
Prognosis	Relatively better	Bad

III- Interstitial cell tumor

These tumors are classified as :

1. Prepubertal tumor (Leydig cell tumor) :

- Characterized by excessive androgen production → sexual precocity

2. Post-pubertal tumor (Sertoli cell tumor) :

- Excessive output of feminizing hormone → gynecomastia, loss of libido & aspermia.

Orchidectomy is curative, in both 1 & 2.

Staging :

Stage I : Tumor in the testis only.
Stage II : Involvement of lymph nodes below the diaphragm.
Stage III : Involvement of lymph nodes above the diaphragm.
Stage IV : Presence of systemic metastases.

Spread :

1. Direct : To testis, epididymis, tunica and skin.
2. Lymphatic : To para-aortic lymph nodes → cysterna chyli → thoracic duct → Virchow's gland.
3. Blood stream : Primary maybe occult with huge nodal or blood secondaries.

Clinical Picture: Various clinical types :

1. Typical :

- Sense of heaviness in testis.
- Painless enlargement of the testis (loss of sensation).
- **On examination :**
  1. The testis is enlarged, hard, smooth, with very early loss of testicular sensation elicited gently to avoid dissemination.
  2. Secondary Hydrocele (10%).
  3. Abdominal exam for the metastasis.
  4. Para-aortic lymph nodes may be palpable just above the umbilicus.
  5. The left supraclavicular region is examined.

> Abdominal mass with an empty scrotum should raise the suspicious of malignant transformation an abdominal un-descended testis

2. Metastatic type :

1. Seminoma : epigastric mass.
2. Teratoma : pulmonary metastasis .

3. Atypical cases : The variations are :

1. Picture of acute epididymo-orchitis.(acute pain & swelling may be due to hemorrhage)
2. The Hurricane type : Fatal in few weeks.
3. The slowly growing type : (teratomatous dermoid cyst).

D.D. :

1. Syphilitic orchitis.
2. Old clotted hematocele.
3. Massive type of bilharziasis of cord and epididymis.

Loss of testicular sensation in :
1.Testicular tumors. 2. Syphilis (gumma). 3. Old hematocele.

Investigations :

1. Tumor markers :

- B-human chorionic gonadotrophin raised in 100% of patients with choriocarcinoma and in less than 10% of seminoma.
- Alpha fetoprotein.*(elevated in Teratoma and never elevated in Seminoma alone)*
- Lactate dehydrogenase.

2. Scrotal ultrasound.
3. CT scan.
4. Plain chest X-ray & abdominal ultrasound.
5. How to get biopsy from a testicular neoplasm ?The scrotum should never be opened to get a biopsy as this may lead to local implantation with subsequent involvement of inguinal lymph nodes. An inguinal incision is made, a vascular clamp is applied to the cord at the deep ring and then the testicle is delivered. A biopsy is taken and frozen section is performed.

Malignant testicular tumour with metastasis in the inguinal lymph nodes :
- **It is a sign of inoperability.** Occurs when :

1. Scrotal skin in infiltrated.
2. After testicular biopsy.
3. After previous orchiopexy.

Management : High simple retrograde orochiectomy through inguinal incision
Scrotal incision is contraindicated

Further management : depending on the type and stage of the disease

1. Seminoma :

Stage I : Radiotherapy for para-aortic lymph nodes.
Stage II : Radiotherapy that extends above the diaphragm
Stage III : Chemotherapy with or without radiotherapy
Stage IV : Chemotherapy

2. Teratoma :

Stage I → Retroperitoneal lymphadenectomy may be done routinely of only if the patient develops lymph node enlargement.
Stage II-IV → Combination chemotherapy.

Prognosis : 5 years survival is :

	Stage I & II	Stage III & IV
Seminoma	95%	75%
Teratoma	85%	65%

Varicocele

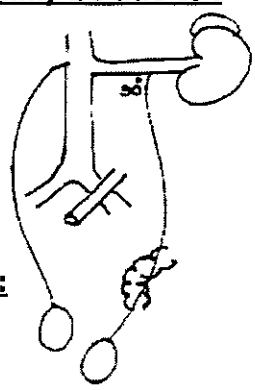
Definition : It is elongation, dilation and tortuosity of the pampiniform plexus veins i.e. varicose veins of the spermatic cord.

Primary varicocele

Etiology :

- **It usually affects young adults (15-30 years & usually on the left side). It may be due to :**

1. Congenial weakness of the wall of the veins.
2. Increased intravenous pressure due to :
   (a) Prolonged standing and sitting.
   (b) Chronic straining and constipation.
   (c) Venous congestion due to unrelieved sexual excitement.



- **Primary varicocele almost always affects the left side, this may be due to :**

1. The right angled termination of the left spermatic vein into the left renal vein.
2. The lower position of he left testis with longer and more dependent plexus.
3. Compression of the left testicular vein by the pelvic colon.
4. Left common iliac vein is crossed by right iliac artery → increase pressure in the vein which transmitted to the pampiniform plexus through the vein of the vas
5. Catecholamines from suprarenal.

Complications :

1. Hypofertility : Varicocele, especially if bilateral can reduce the sperm count or vitality. This due to the higher temperature in the scrotum produced by venous congestion.**(thermal theory)**

- Temperature difference between scrotum and rectum has to be 2.5 degree.
- Even if unilateral, transmission of heat by contact with the other side affects fertility.

2. Repeated attacks of thrombosis.
3. Secondary hydrocele.
4. Testicular atrophy from congestion.
5. Neurosis.

Clinical picture :

- **Usually l5-25 years old**

Symptoms :

- Usually, symptomless and discovered at a medical commission.
- Dragging sensation or aching pain in the testis especially on prolonged standing.
- Picture of complications.

Signs :

- Left testis is at a lower level than the right.
- Soft compressible swelling in the scrotal part of the spermatic cord (bag of warms).
- Thrill is felt on cough.
- The veins empty when the patient lies down and the scrotum is elevated in case of primary varicocele, when the patient stands up again the veins fill from below upwards.
- Abdominal exam to exclude 2ry varicocele.
- **Bow signs :** While holding the varicocele, ask the patient to bow (to bend) :
  1. In primary varicocele : lesion decreases.
  2. In secondary : No effect.

Grading:

- **Grade I:** present only with valsalva.
- **Grade II:** present without valsalva.
- **Grade III:** visible.

Investigation :

1. Doppler and duplex scan: can detect reversal of blood flow in the testicular vein.

Doppler grading :(grading of venous reflux)

- **Grade I:** static
- **Grade II:** intermittent.
- **Grade III:** continous.

2. Exclude cases of secondary varicocele e.g. Hypernephroma.
3. Semen analysis : for cases of infertility.

Treatment :

1. Conservative treatment :

- Reassurance : Avoidance of sexual excitement, avoid constipation . Suspensory belt may be used.

2. Surgical treatment : (see operative book).

- Indications :

i. Oligosperamia : (Sperm count improves in 30-50% of cases after surgery).
ii. A large painful varicocele.
iii. Associated condition necessitating an operation e.g. inguinal hernia or hydrocele.
iv. Failure in medical commission.

Secondary varicocele

The varicosity in pampiniform of plexus of veins is due venous obstruction by pressure upon or invasion of spermatic veins by and abdominal tumor e.g. hypernephroma.

D.D. from primary varicocele :

	Primary varicocele	Secondary varicocele
Onset & duration	Gradual onset and long duration	Sudden onset and short duration
Age	15-25 years	Usually above 40 years
Site	Almost always left	On right or left
Effect on lying down	Disappear	Does not empty
Abd examination	No swelling	Usually reveals renal tumor (hypernephroma)

Hydrocele

Definition : Hydrocele is a collection of clear serous fluid a part of procesus vaginalis.

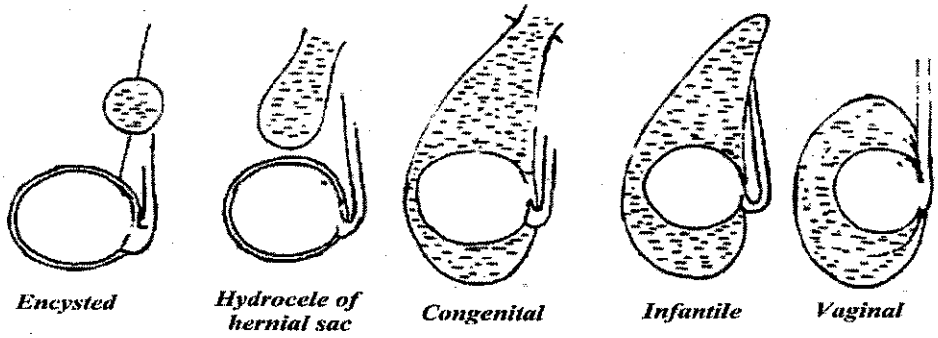
Classification :

1. Hydrocele of the spermatic cord :

(a) Encysted. (b) Hydrocele of a hernial sac.

2. Hydrocele of the tunica vaginalis :

(a) Congenital. (b) Infantile. (c) Vaginal : primary or secondary.



Hydrocele of the spermatic cord

[A] Encysted hydrocele of the cord :

- It is due to persistence of the middle part of the procesus vaginalis.
- There is a smooth, oval swelling which is cystic and translucent.
- By gentle traction upon the testis the swelling moves downwards and becomes less mobile
- **Treatment :** Excision through an inguinal incision.

[B] Hydrocele of a hernial sac :

- Occurs in narrow necked sacs if contents return to abdomen and fail to descend in sac again neck of sac becomes occluded by omentum. Serous fluid collects in sac.
- **Clinically :** History of reducible hernia. Now there is irreducible cystic translucent inguinal or inguinoscrotal swelling.
- **Treated as usual hernia.**

Hydrocele of the tunica vaginalis

[A] Congenital hydrocele :

Is due to : Persistence of the whole processus vaginalis and the communication with the peritoneal cavity is too small to permit the development of a hernia.

Clinical picture :

- A cystic translucent, inguinoscrotal swelling.
- The hydrocele is intermittent, it fills gradually on standing and empties very slowly if patient lies down.

Treatment :

1. Treatment of the cause of fluid in the abdomen (if present).
2. Operation, through an inguinal incision, the sac is divided into two. The upper part is transfixed at the level of the internal ring as a hernia and the lower part is either everted, or is left undisturbed.

[B] Infantile hydrocele :

- The tunica extends up to the internal ring but does not communicate with peritoneal cavity.
- **Treatment :** Eversion of the tunica.

[C] Vaginal hydrocele :

- This is a fluid collection in the tunica vaginalis which may occur spontaneously (primary or idiopathic hydrocele) or may be due to an underlying lesion (secondary hydrocele).

Primary vaginal hydrocele
(Commonest type)

Etiology :

- It occurs in middle aged and elderly males.
- May be due to chronic congestion & irritation by repeated trauma, or diminished fluid absorption by the tunica vaginalis.

The hydrocele fluid :

Is amber yellow, thin and contains water, inorganic salts, fibrinogen and albumin in a concentration that resembles a transudate. In old standing cases, it contains cholesterol.

Complications :

1. Infection leading to pyocele, usually after aspiration.
2. Hemorrhage leading to hematocele either after trauma, or spontaneously.
3. Rupture, usually follows trauma.
4. Hernia of the hydrocele sac due to high pressure inside and a weak point in the coverings of tunica
5. Huge expansion of the scrotum leading to indrawing of the penis which may interfere with micturition and intercourse.

Clinical picture :

- Scrotal swelling (you can get above it) painless and increases in size very slowly.
- The swelling is cystic in consistency (see clinical notes) dull on percussion and by transillumination it is translucent.
- The swelling is irreducible and with no impulse on cough.

- **The testis is identified by :**

1. Palpation at the posterioinferior aspect of the sac.
2. Testicular sensation.
3. Transillumination.
4. Scrotal sonography.

Treatment :

1. Aspiration :

- After transillumination (to localize the testis) the swelling is made tense by manual compression.
- A fine trocar and cannula is inserted into a translucent area and fluid is evacuated.
- This method is indicated when an operation cannot be done e.g. old age.
- Complication :

(a) Recurrence. (b) hemorrhage.
(c) Infection. (d) Injury of the testis.

2. Operative treatment : See operative.

Secondary hydrocele

- **It is secondary to** a disease of the cord or the testis.
- This disease may be **acute** (epididymo-orchitis) or **chronic** (malignant or chronic inflammation).
- It is usually **soft.** It may be **blood stained.**
- **Treatment** is that of the cause.

Hematocele

1. Recent hematocele :

- **Due to :** 1. Injury of blood vessels during aspiration of hydrocele.
  2. Trauma to testis. 3. Blood diseases.
- Swelling becomes painful, tender and not translucent.
- **Treatment :** Urgent operation evacuating the blood and the tunica is excised.

2. Old standing hematocele :

- **Complication :** Infection, atrophy of testis & calcification.
- **Clinical picture :** There is pain and heaviness in the scrotum, together with a swelling, soft (early) or hard (late). It is opaque in transillumination. The contents of the scrotum cannot be differentiated.
- **Treatment :**

1. In early cases, dissection of the clot from the testis and excision of the tunica can be done.
2. In late cases, Orchidectomy is performed to exclude malignancy. It is difficult to differentiate between both unless the testis is bisected.

Cystic Scrotal Swellings

1. Skin : sebaceous cysts.
2. Testes : Cystic teratoma.
3. Epididymis : Spermatocele.
4. Tunica vaginalis : vaginal hydrocele, pyocele, hematocele, chylocele.

D.D. of Scrotal Swellings

1. Testicle : Gumma tumors, acute epididymorchitis.
2. Tunica :

- Hydrocele (translucent) : commonest intrascrotal swelling.
- Hematocele (opaque).
- Pyocele (tender).
- Chylocele (dissolves with ether).

3. Epididymis : T.B.
4. Dartos : Fibroma.
5. Skin : Sebaceous cyst, squamous cell carcinoma.

Epididymal cysts & Spermatocele

Etiology : unknown.

Content : clear fluid. In some cases the cyst contains spermatozoa, which make the fluid turbid, such a cyst is called spermatocele.

Clinical presentation :

- Painless scrotal swelling. The cyst is usually small.
- On examination, the swelling is purely scrotal, lying just above & slightly behind the testis. It is not tender, cystic and translucent (spermatocele is not translucent).
- The main difference from a vaginal hydrocele is that an epididymal cyst is felt separate from the testis.

Treatment :

- **Small cyst :** no treatment.
- **Large progressively growing cyst :** surgical excision.

Pediatric Surgery

Pediatric surgery

General principles

- Infants are very fragile; a child can be become dehydrated from gastroenteritis to the extent of peripheral circulatory failure in a day
- The small baby is easily affected by changes in environmental temperature due to:
  a. Thinner subcutaneous fat.
  b. Inadequate vasoconstriction of cutaneous blood vessels in response to cold.
  c. Inadequate myelination of CNS including heat regulatory center.
  d. Absent of shivering and sweating mechanisms.

Body fluid and Electrolyte

a. The fluid requirement of neonate may be calculated according to the following scheme

	Maintenance IV fluid / day
Neonate	**60**ml/kg to be raised by 10ml/kg daily up to 100 ml
<10 kg	**100**ml/kg
>10 kg	100ml/kg for the 1st 10 kg + **50**ml for each additional kg

b. Daily fluid **maintenance** is given in the formula of 10% glucose in 0.18 percent saline.
c. Potassium chloride (20 mmol/L) is added once urine flow is established.
d. All fluid **losses** are replaced with normal saline with Potassium chloride (20 mmol/L)

Blood volume

a. In neonate, the blood volume is about 80 ml/kg (in adult 70 ml/kg).
e.g. In 3.5 kg neonate will have 280ml total blood volume. Three blood-stained swabs during surgical operation will contain 30 ml of blood which constitute 10% of the total blood volume.

b. For blood transfusion; the following general formula is adequate
10 ml / kg of blood **or** 5 ml / kg of paked RBCs

Respiratory system

a. The lung has sufficient capacity for postnatal survival at about 24 weeks gestation.
b. After delivery, the lung matures rapidly but only about half of the alveoli present at birth.
c. The most energy consuming activity of the neonate is breathing.
d. Respiratory failure develops rapidly in babies with pneumonia.

In case of metabolic acidosis;

Number of mmol HCO3 = Base deficit X WT in Kg / 3

It is a simple formula to estimate the amount of bicarbonate needed.

- Full term babies cannot withstand starvation more than 6h without running the risk of hypoglycemia.
- If the baby cannot tolerate enteral feeding for more than a week, T.P.N is mandatory.
- Morphine is given to adults in a dose of 0.2 mg/ kg, but in the small infant 0.1 mg/kg 6-8h is enough

Common tumors in different age groups

1. **Neonatal period**
   a. Ovarian cyst lesion
   b. Metastasizing neuro-blastoma
   c. Sacro-coccygeal Teratoma
2. **In 1st 2 years of life**
   a. Neuro balstoma.
   b. Nephro balstoma.
   c. Hepato-balstoma.
3. **Between 2-6 years,**
   a. Rabdomyo-sarcoma of the bladder and prostate.
   b. Ovarian Teratoma.
   c. Malignant lymphoma.

Common tumors in children (in order of frequency)

1. Acute lymphocytic leukemia.
2. Brain tumours.
3. Neuro balstoma.
4. Nephro balstoma.
5. Hepato-balstoma.
6. Rabdomyo-sarcoma .
7. Sacro-coccygeal Teratoma

Common features of abdominal tumours in children

1. Abdominal mass.
2. Some tumours present by systemic manifestation as fever, anorexia and failure to thrive.
3. Some tumours present by neurological manifestation as paresis and incontinence.
4. Pain may occur due to meatstasis.

Neuroblastoma

- It is a highly malignant tumour which arises from the sympathetic chain or the adrenal medulla.
- It is reddish purple soft swelling liable to hemorrhage and degeneration.
- It grows rapidly , reaches large size and metastasizes widely by blood and lymph vessels.
- \it is highly radiosensitive but metastasis is so early and widespread that the prognosis is not favorable.